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MALARIA IN AUSTRALIA.

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The distribution of malaria is dependent upon a large range of factors, among which are included (i) all those conditions suitable for the production of large numbers of female Anopheles of varieties which are effective as carriers, (ii) circumstances favourable to the completion of the life cycle of the parasite in the mosquito and (iii) the provision of sufficient human carriers of gametocytes among a population of adequate density.

These three postulates are further modified by geographical conditions of altitude, topography, rainfall and water supply, by variations of temperature, type and distribution of vegetation, by isolation from or contiguity with foci existing among resident populations or nomadic races and by human efforts at prevention and prophylaxis.

In consequence of these numerous factors it is found that, though malaria may be said to prevail approximately from 60° north latitude to 35° south latitude, there are many localities in which one factor or the other prevents the successful endemic establishment of the disease.

Our detailed knowledge of the history of Australia goes back only one hundred and fifty years and in connexion with the study of malaria in that island continent three questions present themselves:

1. What was the situation with regard to malaria among the aborigines immediately prior and subsequent to the coming of the whites?
2. What is the history of malaria in Australia as far as data are procurable?
3. What is the present situation in Australia in regard to the disease?

An attempt will be made to discuss each question in detail.

Malaria Among Aborigines.

It is interesting to speculate as to whether malaria occurred among the natives prior to the arrival of Europeans, but the matter must remain one of speculation only. If malaria did invade the continent, it left no known traces. It is probable that it did not and since both favourable climatic factors and anophelines suitable as carriers are present, the freedom of the people must be referred to a "population" factor. This is undoubtedly the case.

The aborigines were few in number and low in culture. They had no fixed abodes and they did not till the soil, but wandered from place to place over large areas of country, where, speaking generally, native game was comparatively scarce and difficult to kill or capture. Each tribe had as little contact with each other tribe as was possible; residence in camps was rarely sufficiently long for the development and transfer of malarial parasites and on those rare occasions when the aborigines of the north of Australia made more permanent camps in the wet season, these were deserted as soon as the rains began to lighten, that is to say, before the time which favours the spread of malaria in Northern Australia.

Under these circumstances it is not to be wondered at that no evidence of the disease has been recorded.

That their freedom from disease was not the result of any immunity or partial immunity was evidenced when the aborigines abandoned their old customs and came to live with and work for the white man, for they readily contracted malaria and occasionally acted as reservoirs for the dissemination of the parasite. It is probable, indeed, that by their agency the disease was spread during the pioneer days of north Australia to those big cattle stations and mining fields on which aborigines were employed (see map: 18, 19, 20, 21, 22, 23).

Several small foci of endemic infestation have been discovered among aborigines from time to time, for example, in some of the Torres Straits Islands, in Palm Islands, north Queensland (see map: 3) and Melville Island, Territory of North Australia (see map: 18, 22); others are suspected, as in the Gulf of Carpentaria and Cape York Peninsula, but in general they have been and remain free from the disease.

Malaria in Australia Among Whites.

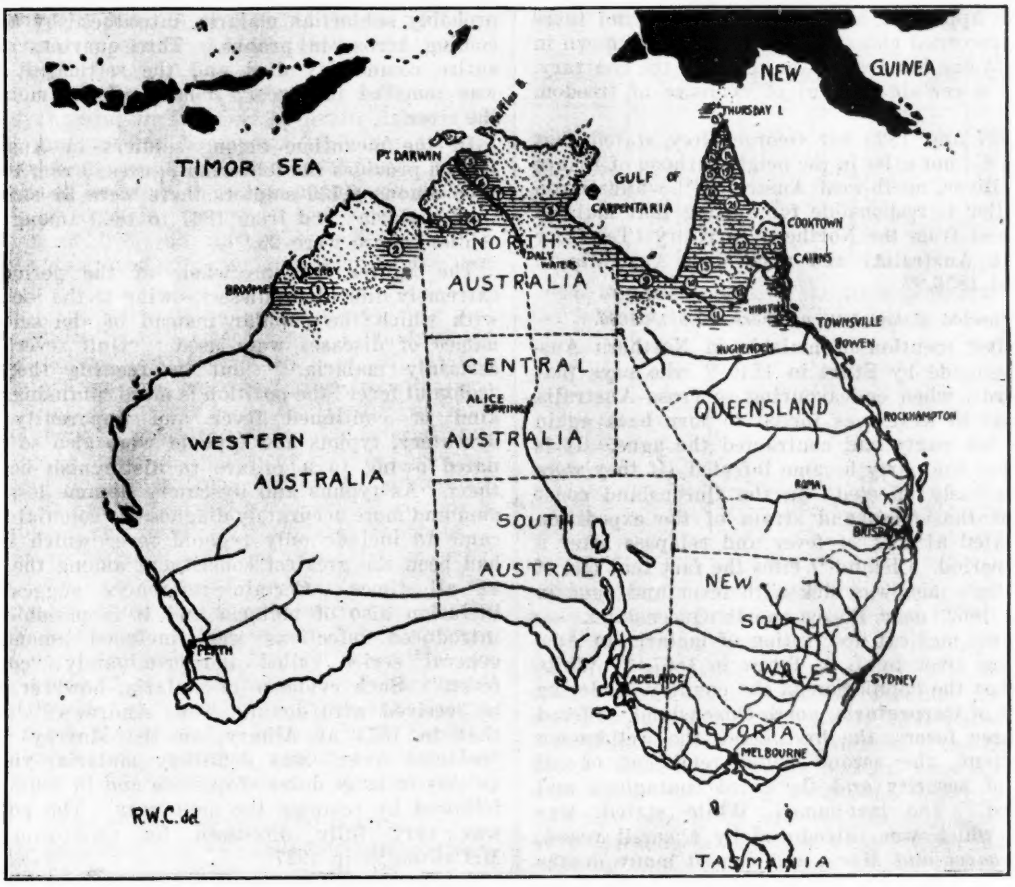
The first reference that can be found to malaria in Australia is that by Jackson[1] who states that the hospital at Moreton Bay (now Brisbane, the capital of Queensland), which was opened in 1825, showed in the twelve months, December, 1827, to December, 1828, among 956 patients admitted for all causes, 27 recorded as suffering from "intermittent fever" and 161 illnesses recorded under the name "*febris*." Among the 27 illnesses recorded as intermittent fever, probably there were included conditions other than malaria. In the year 1832 in the month of January there were in the same settlement 127 cases of intermittent fever, mainly from Brisbane and Eagle Farm (four miles from Brisbane) and in all for the same year there were 665 cases of intermittent fever recorded, of which four were fatal. An estimation of the population of the settlement made in October, 1832, showed that it consisted of 838 men, 59 women and 66 children and of this number 782 were convicts; the remainder belonged mainly to the 17th Regiment.

If the disease recorded was malaria, and it appears to have been so in some cases, it is not likely that the convicts brought it to Moreton Bay. It is much more probable that it was introduced by some of the officers and men of the regiment. In this connexion Lieutenant Breton remarks that Captain C., of the 17th Regiment, is responsible for the following statement:[2]

> I am, however, far from thinking our present place of abode unhealthy, although fever and ague have of late been rather prevalent; but these were not known when

first I came here and probably will not always continue to annoy us; it is strange that during the last very hot weather, when these complaints were most prevalent, the soldiers suffered more than the convicts; but such a circumstance never occurred before or since.

While it would seem from these accounts that malaria was successfully introduced to Moreton Bay (Brisbane) in 1828 and 1832, it does not appear



DISTRIBUTION OF MALARIA IN AUSTRALIA.

The areas in black are intensely malarious; the shaded areas indicate endemic areas in Australia and the stippled areas localities where malaria is presumed to be endemic.
..... = Railway line under construction, approved or surveyed.
———— = Main railway lines in active operation.

With the exception of the two transcontinental railways from South Australia north and west, the distribution of the railway lines gives a fair guide to the distribution of population. It will be observed that population is sparse as yet in the malarious areas.

The numerals indicate areas where cases have occurred recently, apart from Broome, Derby and Cairns which are mentioned by name. During the compilation of this statement a small epidemic occurred at Poid Village near Thursday Island with 98 cases and several deaths. 1 Fitzroy River Valley, 2 Ord River Valley, 3 Victoria River Valley, 4 Willeroo, 5 Port Darwin area, 6 Pine Creek and Umbrawarra area, 7 Katherine River area, 8 Roper River area, 9 Burketown area, 10 Normanton area, 11 Croydon area, 12 Forest Home-Einasleigh-Kidston area, 13 Van Rook area, 14 Kendall River area, 15 Aurukun-Rockeby area, 16 Weipa-Batavia River area, 19 Merluna-Mein area, 20 Coen-Moojeeba area, 21 Ebagoola-Yarraden-Musgrave area, 22 Maytown-Palmer River area, 23 Yarrabah-Cairns area, 24 Cairns-Innisfail-Mourilyan area (rare), 25 Cardwell-Halifax-Ingham area (rare).

that it existed, at any rate in endemic form, at Raffles Bay in the Territory of North Australia. Dr. T. B. Wilson[3] mentions that Dr. R. M. Davis in his annual medical report from June, 1828, to June, 1829, states:

Nothing in the form of epidemic or contagious disease has been observed. The greater portion of the diseases which have occurred are to be attributed to the want of a due quantity of vegetable food . . . The people, though generally complaining of not possessing as much bodily strength as heretofore, look more healthy than can be at first imagined. The health of some men, delicate on arrival here, has much improved . . . No diseases have occurred within the year . . . Although in the summer months the atmosphere is very hot and at times oppressive, few cases of fever or other acute disease at that period prevailed, even among those men who were obliged to be much exposed to the sun's influence . . . Captain Laws also supports the healthiness of this part, stating that the principal disease seems to have been scurvy, which they suffered from soon after their arrival.

Dr. Wilson mentions, however, the arrival of the *Amity* at Raffles Bay on June 31, 1829, two men

from her dying from fever on July 2. Their fever was acquired at Koepang (Timor) and was almost certainly malaria.

It would appear, therefore, that as early as 1829 the possibility of the introduction of malaria into Australia occurred also at Raffles Bay. As at Moreton Bay, however (and these are the only two instances found), the introduction of the disease does not appear to have been successful and there was no recorded endemic area of malaria known in Australia one hundred years ago. On the contrary, there is a certain amount of evidence of freedom from it.

In 1837 and 1838 Sir George Grey stated that malaria did not exist in the neighbourhood of Prince Regent River, north-west Australia,[4] while Baron von Müller is responsible for stating that malaria was absent from the Northern Territory (Territory of North Australia) and north-west Australia in 1855 and 1856.[5]

Reported Malarial Occurrences, 1849 to 1880.

The first mention of malaria in Northern Australia is made by Sturt in 1849[6] who says that Leichhardt, when endeavouring to cross Australia from east to west, was forced to turn back again because his party had contracted the ague. It is likely that the party became infected (if they were not previously affected) on the Queensland coast and that the stress and strain of the expedition precipitated attacks of fever and relapses after a certain period. Cleland[7] cites the fact that one of MacKinley's men was sick with fever and ague in August, 1862, near Bowen, north Queensland.

The first medical description of malaria in Australia was given by J. A. White in 1867.[8] White states that the population in the country bordering the Gulf of Carpentaria, north Queensland, suffered from three fevers, the first being the well-known intermittent, the second being remittent of all grades of severity and the third contagious and continued. The last-named, White stated, was typhoid which was introduced by a small vessel, the *Margaret and Mary*, and caused many deaths in the gulf area. The intermittent fever, of course, was malaria. The remittent type is of great interest. The symptoms are given by White as being as follows:

> There was a very sudden onset, 11 a.m. to 2 or 3 p.m., with a dizziness, slight chills and flushes, wandering pains in the extremities, a fixed pain in the back and epigastrium, with an agonizing pain in the frontal and supraorbital regions. Nausea existed and towards evening a quantity, generally small, of a dark yellow, acrid, intensely bitter fluid, having a foul odour, was discharged from the stomach. The pulse was generally 100, full and strong. The lateral margin of the tongue was purplish, but covered with whitish or slight yellow fur. The patient was delirious and generally unable to sleep. Towards 2 or 3 a.m. the symptoms abated, but did not disappear and another paroxysm commenced at about 10 a.m., being generally more severe. In cases of recovery the fifth such paroxysm was the mildest and last. The treatment given consisted of a purge and six grains of quinine every four hours. Each paroxysm consisted of a hot stage, which might, or might not, be preceded by rigors or chills; sweating sometimes did, and sometimes did not, occur after the hot stage.

This almost exactly describes the type of subtertian malaria so common in the neighbouring parts of Melanesia and it is interesting in that connexion, since the coastal area of the Gulf of Carpentaria was often visited by people who were trading throughout Melanesia.

In the year 1864 the population of Burketown was almost wiped out by the "black sickness," probably subtertian malaria, introduced by Malays coming across in prahus. Three-quarters of the entire community died and the settlement itself was removed to Sweer's Island off the mouth of the river.

In the meantime among soldiers in Australia Hirsch provides the following figures: From 1859 to 1866 among 6,736 soldiers there were 31 suffering from malaria; and from 1867 to 1869 among 4,491 soldiers there were 25.[9]

The figures for the whole of the period are extremely difficult to dissect, owing to the looseness with which the popular instead of the scientific names of diseases were used. "Gulf fever" was certainly malaria,[10] but as regards the term "colonial fever" the position is more confusing. Any kind of continued fever and apparently true dysentery, typhus and typhoid were also so designated owing to a failure to distinguish between them. As typhus and dysentery became less common and more accurately diagnosed, "colonial fever" came to include only typhoid fever which indeed had been the greatest constituent among the cases at all times. Certain references suggest the inclusion also of malaria and it is possible that introduced infections were included among the general series called indiscriminately "colonial fever." Such evidence of malaria, however, must be received with doubt. Thus Andrews[11] stated that in 1874 at Albury, on the Murray River, "colonial fever" was definitely malaria, yielding quickly to large doses of quinine and in some cases followed by relapses the next year. The position was very fully discussed by Cumpston and McCallum[12] in 1927.

The Period of Greatest Frequency, 1880 to 1890.

Up to 1880, therefore, it would appear that malaria was occasionally introduced both in the southerly and the most northerly parts of the continent and made a doubtful local headway, but that it failed to establish itself except in the area around the Gulf of Carpentaria, where it became endemic.

Undoubtedly the sparsity of population contributed very largely to this fortunate freedom from endemicity. In 1880 to 1890 there were temporary concentrations of population in relation to mining activities in many parts of Australia and the third factor in the causation of the disease now being favourable, malaria reached its height so far as its history in Australia is concerned. It became endemic through a large part of Queensland, the Territory of North Australia (Northern Territory) and north-west Australia and in addition small local epidemics frequently occurred.

Thus Holmes[13] states that malaria was very prevalent on the Pine Creek goldfield and railway construction work in the Northern Territory in 1879 to 1883 and that the aborigines at Melville Island, Northern Territory, suffered from subtertian outbreaks with considerable mortality.

During the opening up of the goldfields in the Northern Territory from 1879 to 1883, figures for the first year show 61 out of 166 deaths as being due to malaria, 61 out of 154 in 1880 and 51 out of 100 in 1881.[13]

Again, in 1884 Jee stated that malaria was formerly present in the Torres Straits Islands,[14] but the statement of Browne in 1885 that malaria was extremely common in Charters Towers (north Queensland) and that the whole population suffered from it, is probably incorrect and relates to an extension of the epidemic of dengue present in Rockhampton and the neighbourhood at that time.[15][16]

In this connexion it is interesting to note that at Hughenden (north Queensland), further in the interior, during 1886 to 1890 26% of all patients admitted to hospital were suffering from malaria and a point of interest made by Hunt[17] is that, whereas the type was formerly frankly intermittent, it had by 1890 become remittent in type, probably indicating a change from tertian to subtertian, which is the reverse of the usual procedure, but is explicable if one recollects that large numbers of natives from malarial countries were being imported at this time to work sugar.

Heydon[18] states from the literature that in 1883 malaria was very prevalent at Yarrabah and Cairns, north Queensland. In 1889 Dyson[19] says that it occurred at Normanton, the Palmer goldfields, Croydon, the Johnstone River and Cairns, all of north Queensland. Indeed, it is reported from almost every area of virgin country then under process of development and settlement.

Period of Initial Decline, 1891 *to* 1900.

In the following decade (1891-1900) a considerable change for the better occurred. It may be indicated by the following figures for Hughenden supplied by Hunt.[20] The frequency of admissions to hospital for malaria were as follows:

1887	35·20%
1888	30·10%
1889	20·90%
1890	15·06%
1891	19·10%
1892	8·50%

Other areas reflected the same general trend. Ahearne[21] says that the disease, though previously frequent, was rare in Townsville (north Queensland) in 1890. Jefferis Turner[16] in 1890 said that in fifteen months in Brisbane (south Queensland) he had seen only three infected children and that one of these came from the north. In 1900 malaria was still fairly common in Cairns according to O'Brien, but in 1905-1908 there was none recorded.[22]

While this general improvement affected the greater part of Queensland and while malaria as a disease ceased to exist in the southern States, the position in the Northern Territory from 1890 to 1900 did not improve at the same rate. The number of deaths due to fever in the Northern Territory over a period of forty years among a population varying between 3,000 and 4,000 individuals of mixed races was 412.

Year.	Deaths.	Year.	Deaths.	Year.	Deaths.	Year.	Deaths.	Year.	Deaths.
1871	—	1879	61	1887	4	1895	3	1903	6
1872	1	1880	61	1888	5	1896	7	1904	8
1873	13	1881	51	1889	6	1897	8	1905	—
1874	7	1882	9	1890	6	1898	5	1906	—
1875	15	1883	23	1891	—	1899	6	1907	7
1876	3	1884	4	1892	2	1900	9	1908	16
1877	1	1885	3	1893	3	1901	6	1909	18
1878	7	1886	2	1894	5	1902	6	1910	18

The admission book at the Darwin Hospital gives the following figures:

Year.	Admissions.		Year.	Admissions.		Year.	Admissions.	
	Malaria.	Total.		Malaria.	Total.		Malaria.	Total.
1897	18	77	1902	1	65	1907	12	114
1898	8	80	1903	2	50	1908	23	136
1899	6	68	1904	12	89	1909	44	129
1900	5	65	1905	1	74	1910	27	165
1901	1	59	1906	6	113			

Thus for fourteen years there is a total of 166 cases. These figures are supplied by Breinl.[23]

As has generally been the case, the high figures for the years 1908, 1909 and 1910 are associated with concentration of population due to mining activities, a payable field having been discovered at Umbrawarra Creek, ten miles from Pine Creek, in 1909. Wherever payable mineral was discovered and where the living conditions were poor and mosquito infestation considerable and where many were attracted among the miners from malarious parts of Queensland, New Guinea and Western Australia, the disease showed itself at once and was usually severe. In Umbrawarra, for example, the living conditions were bad, the men camped on the banks of the creek among excellent mosquito breeding places, numerous large square holes were excavated on each side of the creek by miners searching for alluvial tin and the miners who were poorly fed, worked up to their hips in water a large part of the time.[23]

Continued Decline, 1901 *to* 1929.

The decade 1901-1910 saw another change for the better in Queensland. The Kanakas had been repatriated, mining had become more stabilized and living conditions were better.

The disease was still bad in north-west Australia at Wyndham in 1903 according to Belgrave[24] and was still occurring along the east coast of north Queensland from 1905 to 1908 according to O'Brien,[22] while in 1910 it recurred in and around

Cairns (north Queensland), though the type was more benign.[25]

An epidemic of explosive violence occurred in Queensland in 1910 and Elkington[26] reports on this outbreak as follows:

> The outbreak occurred at Kidston on the Einasleigh goldfield, north Queensland. The population was 400. Two miners from New Guinea with subtertian infection inoculated the local mosquitoes. Before the outbreak subsided in this population of 400 there were 120 cases with 24 deaths.

In the same year Breinl reported that a few cases were still occurring in Cairns, Cooktown and the gulf country of north Queensland. The type was benign tertian, except for a few malignant cases from New Guinea.[27]

In the opening half of the decade 1911-1920 the improvement still continued, but in the later period a large number of soldiers infected with malaria returned to Australia from New Guinea, Palestine and other malarious war areas. According to figures given later this influenced very little the total number of deaths reported for the Commonwealth. It did, however, reflect itself because of the relapses in the statistics of those States in which malaria was a notifiable disease. The figures for Western Australia will serve to illustrate this.

Year.	Cases of Malaria Notified.	Remarks.
1913	41	None in south of State.
1914	19	None in south of State.
1915	2	
1917	13	
1918	8	
1919	686	680 among the military.
1920	57	16 from Wyndham.
1921	39	20 from Wyndham. 6 from Derby. 2 from Broome.
1922	41	22 from Wyndham. 5 from Broome.
1923	23	
1924	29	Mainly imported or from the north.
1925	26	
1926	18	

The notifications are back to prewar numbers again now.

The possible danger to Australia was realized and a conference met in Sydney and drew up resolutions for the protection of the Commonwealth.[28] These resolutions may be summarized as follows:

> (i) Returning soldiers from New Guinea infected with malaria were to disembark at Townsville, north Queensland, for treatment;
>
> (ii) Invalided and discharged soldiers who had suffered from malaria, were to be collected in one place for treatment;
>
> (iii) Military pay was to continue during treatment;
>
> (iv) The treatment officially recommended was to be used.

On June 3, 1916, the first three of the resolutions were put into force.[29]

In addition to these measures the Commonwealth Government ordered a survey of the Murray River irrigation areas which had been selected for the settlement of returned men. This was carried out by Taylor from October, 1916, to January, 1917. Despite rather unfavourable weather conditions, he found anophelines breeding in most of the areas visited and in all he found conditions favourable to anopheline propagation.[30]

During the present decade (1921-1929) malaria has reached a figure so low that the deaths attributable to the disease, about five per million, are not likely to be permanently reduced. The fairly constant malarial level is revealed by the deaths attributed to the disease during the last twenty-three years.[31]

Year.	Deaths.	Year.	Deaths.	Year.	Deaths.	Year.	Deaths.
1905	55	1911	19	1917	49	1923	21
1906	38	1912	17	1918	41	1924	20
1907	42	1913	24	1919	34	1925	35
1908	52	1914	22	1920	19	1926	25
1909	59	1915	30	1921	43	1927	29
1910	55	1916	50	1922	21		

The increased deaths in 1916, 1917 and 1918 due to returned soldiers will be noted.

The Australasian Medical Congress held at Brisbane in 1920 passed a resolution asking that the activities of the Australian Hookworm Campaign, a body financed by the Commonwealth of Australia, the International Health Board of the Rockefeller Foundation and the Government of the State of the Commonwealth in which the hookworm campaign was working, be extended to include a survey of Australia for malaria and filariasis.[32] The difficulties in the way of this undertaking were considerable and the figures obtained can be regarded only as the most general of indications, the positive being of considerably greater value than the negative findings. Of 9,022 people examined in Western Australia, the Northern Territory and north and south Queensland, 44 were found to harbour parasites in the blood and these were distributed mainly between Cairns, Bowen to Townsville, Nambour to Maryborough (all of north Queensland); three cases were detected in the Brisbane-Southport area, which is not an endemic locality, one was found in north-west Queensland and four in the Northern Territory.

Notifications of malaria which are compulsory in all States but one in Australia, are very unsatisfactory. In the last twenty years there have been at least ten alterations in the number of States notifying malaria; at times only one State has demanded it; in one year all States did so. Moreover, when malaria is mild, a medical practitioner is not sent for; again, the disease is often unrecognized, while notification is frequently omitted. Further, the large sale of tonics containing quinine which occurs in malarious areas, tends to mask the effect of the disease in those communities.

From time to time isolated cases have arisen in parts of Australia which are not ordinarily regarded as malarious. Thus in New South Wales Jamieson reported a case contracted at Gosford in 1915,[33] Evans reported a case contracted at Wyong in 1919,[34] Clayton and Utz reported a case contracted near Wagga in 1921[35] and Money reported a case

contracted at Sydney in 1926.[36] In Western Australia Baldwin reported a case arising at Maylands near Perth in 1920.[37]

The Present Situation With Regard to Malaria.

Australia is set in immediate relationship to one of the most malarious areas in the world. All forms of the disease flourish in the great island of New Guinea and the New Guinea archipelago which include between them the Territory of Papua and the Mandated Territory of New Guinea. The disease is severe in the British Solomon Islands protectorate and in most parts of the condominium of the New Hebrides; that is to say, throughout almost the whole of the great Melanesian arch which runs parallel to the northern and north-easterly coasts of Queensland. Its distribution is abruptly terminated easterly by the 160th meridian of east longitude and southerly by the 20th parallel of south latitude. West and south of these two lines the Austral-Pacific zone is quite free from malaria, a freedom based upon the absence of suitable anophelines to act as vectors. North of 20° south latitude and west of 160° east longitude the situation is very different, every area up to the equator being very heavily infected with the exception of Australia.

In Australia at the present time malaria is confined to a small area in north-west Australia, to two areas of population in the Territory of North Australia (Northern Territory) and to two areas in north Queensland and in all these localities with the exception of Cairns the infection is almost solely confined to cattle-men, miners, sugar cane labourers and aborigines. In all instances again these are men who often live and work under very primitive conditions, with poor food and lodging.

In the Northern Territory malaria is found on the two watersheds, that is to say, along the rivers running into the Timor Sea and along those running into the Gulf of Carpentaria. Settlement is exceedingly sparse, but were it closer, it is probable that malaria would be much more widely disseminated.

In north Queensland malaria exists along the shores of the Gulf of Carpentaria and in the neighbourhood of Cairns and to the north through Cape York Peninsula. In this large area there are several aboriginal mission stations where numbers of aborigines are congregated and it is known that malaria has existed in the past among them and is still endemic.[38][18]

The history of malaria in relation to the township of Cairns, the only large town implicated, is interesting and important. As has been said, there have been several epidemics of considerable intensity and the area is still an endemic centre, though its virulence has decreased. In 1918 the disease was widespread and a serious handicap to development, as established by the survey of Breinl and Taylor who found that 13·5% of the specimens examined contained malaria parasites. Since that date much progress has been made in the general sanitation of the town; in fact, the Commonwealth Health Laboratory now stands on what was a notorious chain-wide swamp, a proved breeding ground of *Anopheles annulipes* and *Anopheles bancrofti*. Extensive swamps still exist on the outskirts of the town and as recently as October, 1928, an outbreak of malaria occurred among the inhabitants around the Draper Street swamp, a breeding place also incriminated in 1918. During the past nine months fifty blood films from white persons have been examined and malarial parasites detected in five. The youngest patient was three months of age, the mother having had malaria before the birth of the child; one child, aged five years, died in hospital with cerebral symptoms and cachexia, while the remaining three patients were adults who volunteered information as to other members of their families with fever, from whom, however, specimens could not be obtained. Seventy specimens taken at random from coloured people with the object of determining whether the people were carriers or not, were found in all instances to be free from parasites, but specimens taken from three Malays visiting Cairns in pearling luggers from further north, were all found heavily infected with malarial parasites. The intercourse between this type of native and the population in relation to the native quarter of Cairns, where hookworm infection, overcrowding and swampy and dirty conditions generally prevail, probably represents the only important factor in the present endemicity. It must be recollected, of course, that the habit of taking quinine for any febrile disturbance, which is very general throughout north Queensland, greatly reduces the finding of parasites in films examined. It would seem at present that small outbreaks may be expected, but if the progress of the past ten years be continued, Cairns should soon be practically free from malaria.

During the last twelve months there were in all eight deaths from malaria in Queensland, but generally the patients now treated in hospital, elsewhere than in north Queensland, are visitors from the Territory of New Guinea, the Territory of Papua and from the other islands of the Melanesian chain. Australia does a large trade with these infected areas and the white people living there as traders, planters and so forth are largely recruited from Australia. There is also an increasing number of people from India and the Straits Settlements who make a short holiday trip to Australia or New Zealand to save portion of the time that would otherwise be spent on the long trip to England.

The distribution of anopheline mosquitoes throughout Australia is relatively heavy and although there still remains much work to be done in connexion with the distribution of these insects, the following facts have been determined. The anopheles represented in Australia are as follows: *Anopheles annulipes, Anopheles annulipes* var. *amictus, Anopheles bancrofti, Anopheles punctulatus, Anopheles punctulatus* var. *moluccensis, Anopheles atratipes, Anopheles stigmaticus.*

There is no scientific proof as to the efficiency of any but two of these varieties as malarial vectors.[39] Heydon[40] has shown that in New Guinea *Anopheles punctulatus* and *Anopheles punctulatus* var. *moluccensis* are efficient carriers. From recent private correspondence it would appear that malaria used therapeutically has been conveyed by *Anopheles annulipes*. At the present time the workers at the Australian Institute of Tropical Medicine at Townsville, north Queensland, are investigating the ability of *Anopheles bancrofti* to act as an efficient carrier, as previously suggested by Maplestone.[41]

Conclusion.

Since the distribution of *Anopheles annulipes* is general throughout the continent and since there have been large influxes of infected persons into areas where the vector not only existed, but where meteorological conditions were favourable for the spread of the disease, it is pertinent to inquire why malaria has as yet been successfully implanted only in a few small localities and why it does not flourish even in these.

Ross[42] discusses the factors necessary for the spread of the disease, emphasizing the importance of the number of anophelines, the number of human beings, the number of human beings with malaria and the character of the human population. He also discusses the question of the slow change in the malaria state of large areas and instances the increase in Mauritius and the disappearance in England.

To these might be now added the recent explosive outbreak and subsequent endemicity in Barbadoes, a locality free for centuries and believed immune on account of the fish which inhabit the fresh waters of its areas.

Ross discusses the factors suggested for the disappearance of malaria from England, namely, (i) the reduction of anophelines by drainage, (ii) the reduction of population by emigration, (iii) the use of quinine. He believes that the first factor is all important and the last relatively important.

It has been shown that in Australia there is no suitable native population to act as a reservoir of virus. It must also be recognized that the population in general is very sparse in those areas reputed to be malarious, but it is not considered that these two factors are alone sufficient to account for the absence of the disease.

On many occasions there has been a congregation of human carriers, a sufficiency of suitable mosquitoes and meteorological conditions suitable for the spread of the disease, yet the latter has spread on some occasions and not on others. A more careful consideration of the data reveals the fact that malaria has spread when conditions for the white population have been primitive, where hardship is rife, where sanitation is poor and where work is heavy. In this connexion the remark of Sir Malcolm Watson may be recollected, that "the poor are malarious and the malarious poor."

Outbreaks in Australia have occurred principally on mining fields and upon cattle stations. In the latter localities, no doubt, the factor of a native reservoir of infection must also be conceded. On the other hand, where conditions have been better in soldier settlements and largely in sugar-growing areas, malaria has died out. It does not seem to be due to any lack of numbers in the anopheline population. No deliberate attempt has been made in the direction of drainage for mosquitoes and farm drainage in general is not well advanced.

A factor which deserves consideration, is the presence of domestic animals in agricultural areas. Recent French and Italian work shows that anophelines often prefer such a blood supply, thus diverting these insects from man.

It is interesting to note that in other parts of the world the importance of the economic factor has been recognized. Thus Marchoux[43] compares two rural areas in the Rhône Valley. In both malaria was formerly a scourge and in both *Anopheles maculipennis* is abundant at the present day. In one, however, Les Dombes, malaria no longer exists. In this district the peasantry were formerly sunk in extreme poverty, underfed and overworked. Drainage of more than half the area of the anopheline breeding grounds during the first half of the nineteenth century produced little effect on malaria. During the latter half of the century, however, the conditions of the peasantry vastly improved; they were no longer overworked, underfed and oppressed by care; the number of cattle increased. Coincident with these changes malaria decreased and finally disappeared, although some of the drained breeding grounds have been restored. In the second district, La Camargue, although *Anopheles maculipennis* is less abundant than in Les Dombes, malaria is still prevalent; but here the labourer is miserably lodged and badly fed.

Seeing that the mosquitoes are still prevalent in both these areas, it is in the economic factor we must seek our explanation of the phenomena above. Drainage leads to better land, to more animals, to better houses and a higher economic status.

It would seem that in Australia the all-important factor is the economic and hygienic status of the people.

It is interesting to speculate as to whether this is due to increased bodily resistance, to less opportunity for mosquitoes biting or to some difference in the time of appearance or the absolute numbers of malarial gametes in the blood. There is, however, nothing at present to guide one in choosing the factor or factors concerned in this relative immunity.

Minor factors which have influenced the decline of malaria in Australia, are the diminution of the numbers of the aborigines and their smaller and smaller contact with Europeans; again, there has been the policy which led to the deportation of the Kanaka workers in the Queensland sugar cane fields and prevents the admission of further coloured

labourers of lower hygienic status and with poorer standards of living.

Of supreme importance, however, has been the high economic standard of living enjoyed by most workers in the tropical and subtropical areas, a condition of the happiest augury for the future and one which, combined with watchful care on the part of the Commonwealth Department of Health, bids fair to safeguard Australia from a menace that has crippled many another country.

References.

(1) E. S. Jackson: "The Malaria Danger," THE MEDICAL JOURNAL OF AUSTRALIA, June 25, 1921, page 527.

(2) Breton: "Excursion in New South Wales," 1833 (cited by J. B. Cleland).(7)

(3) R. M. Davis: "Narrative of a Voyage Round the World," 1835 (cited by T. B. Wilson).

(4) G. Grey: "Journal of Two Expeditions of Discovery in North-West and Western Australia" (cited by J. B. Cleland).(7)

(5) F. von Müller: Howitt's "History of Discovery in Australia," Volume II.

(6) Sturt: "Expedition into Central Australia (1849)," Volume II.

(7) J. B. Cleland: "Contributions to the History of Disease in Man in Australia," Annual Report, Department of Public Health of New South Wales, 1912.

(8) J. A. White: "On the Fevers of the Gulf of Carpentaria," *Australian Medical Journal*, 1867, Volume XII, page 361.

(9) A. Hirsch: "Handbook of Geographical and Historical Pathology," 1883, Volume I.

(10) P. James: "Remarks on the Fevers and Diseases of Tropical Queensland," *Australasian Medical Gazette*, July, 1891, page 300.

(11) A. Andrews: "Typhoid and Colonial Fever," *Australasian Medical Gazette*, 1911, page 351.

(12) J. H. L. Cumpston and F. McCallum: "The History of the Intestinal Infections (and Typhus Fever) in Australia, 1788-1923," Service Publication No. 36, Commonwealth Department of Health.

(13) M. J. Holmes: *Bulletin of the Northern Territory*, May, 1913, Health report for the year 1912.

(14) H. C. Jee: "A Few Remarks on Torres Straits," *Australasian Medical Gazette*, 1884, page 223.

(15) G. Browne: "Charters Towers from 1882 to 1890," *Australasian Medical Gazette*, September, 1890, page 319.

(16) A. J. Turner: "On Disease Among Children in Brisbane," *Australasian Medical Gazette*, 1891, page 65.

(17) J. S. Hunt: "The Evolution of Malaria," *Australasian Medical Gazette*, December, 1890, page 75.

(18) G. M. Heydon: "Report of Investigation into Malaria and Filariasis in Cairns and Elsewhere," *Health*, September, 1927, page 133.

(19) T. S. Dyson: "Malarial Fevers of Tropical Queensland," *Transactions of the Second Session of the International Medical Congress of Australasia*, 1889, page 64.

(20) J. S. Hunt: "Notes on the Demography of North Queensland," *Transactions of the Third Session, Intercolonial Medical Congress*, 1892, page 598.

(21) J. Ahearne: President's Address of the North Queensland Medical Society, *Australasian Medical Gazette*, August, 1890, page 293.

(22) R. A. O'Brien: "Notes from North Queensland," *Australasian Medical Gazette*, March, 1908, page 121.

(23) A. Breinl: "Report on Health and Disease in the Northern Territory," *Bulletin of the Northern Territory*, March, 1912, page 35.

(24) T. B. Belgrave: "The Pathogenesis and Prophylaxis of Typhoid Fever with some Observations in the Same Connection on Malaria in the North-West," *Australasian Medical Gazette*, February, 1903, page 60.

(25) R. A. O'Brien: "Ankylostomiasis and Other Tropical Diseases in Queensland," *Transactions of the Eighth Session, Australasian Medical Congress*, 1908, Volume II, page 326.

(26) J. S. C. Elkington: "Quarantine in Queensland," *Australasian Medical Gazette*, April 27, 1912, page 433.

(27) A. Breinl: "Report of the Australian Institute of Tropical Medicine for the Year 1910," page 21.

(28) A. Breinl: "The Malaria Problem," THE MEDICAL JOURNAL OF AUSTRALIA, January 29, 1916, page 106.

(29) A. Breinl: "Malaria in Returned Soldiers," THE MEDICAL JOURNAL OF AUSTRALIA, June 3, 1916, page 453.

(30) F. H. Taylor: "Malaria Mosquito Survey of Irrigation Areas in the Murray River District," Service Publication No. 12, Commonwealth Department of Public Health.

(31) "Commonwealth Year Book," Numbers 3 to 21, 1909-1928.

(32) "Report of Sub-Committee on Tropical Australia," *Transactions of Eleventh Session of the Australasian Medical Congress*, 1920, page 39.

(33) S. Jamieson: "Malaria Arising in a Non-Malarial District," THE MEDICAL JOURNAL OF AUSTRALIA, February 20, 1915, page 163.

(34) W. Evans: "Anti-Malarial Works with the Australian Mounted Division in Palestine," THE MEDICAL JOURNAL OF AUSTRALIA, December 20, 1919, page 526.

(35) J. H. Clayton and L. Utz: "A Case of Malaria Infection in the Riverina, New South Wales," THE MEDICAL JOURNAL OF AUSTRALIA, May 7, 1921, page 382.

(36) R. A. Money: "A Case of Malaria Acquired in Sydney," THE MEDICAL JOURNAL OF AUSTRALIA, August 28, 1926, page 283.

(37) A. H. Baldwin: "An Investigation into Hookworm Disease, Malaria and Filaria in Western Australia," Report of the Australian Hookworm Campaign Survey, 1922.

(38) "Report of the Australian Hookworm Campaign."

(39) G. F. Hill: "The Distribution of Anopheline Mosquitoes in the Australian Region," *Proceedings of the Royal Society of Victoria*, 1925, Volume XXXVII, page 61.

(40) G. M. Heydon: "Malaria at Rabaul," *Health*, November, 1923, page 249.

(41) P. A. Maplestone: "Malaria in Australia," *Annals of Tropical Medicine and Parasitology*, 1923, Volume XVII, page 213.

(42) R. Ross: "The Prevention of Malaria," 1910.

(43) E. Marchoux: "*Le Paludisme dans les Dombes et en Camargue*," *Bulletin de l'Académie de Médecine*, January 11, 1927, page 67.

Bibliography.

Annual Reports of Department of Public Health of Western Australia, 1913, 1914, 1915, 1916, 1918, 1919, 1920, 1921, 1922, 1923, 1924, 1925, 1926.

A. Breinl: "The Object and Scope of Tropical Medicine in Australia," *Transactions of the Ninth Session of the Australasian Medical Congress*, 1911, Volume I, page 527.

A. Breinl: Reports of the Australian Institute of Tropical Medicine, January-June, 1914, January-June, 1915, July-December, 1915, January-June, 1916, July-December, 1916, January-June, 1917, July-December, 1917, July-December, 1918, Yearly Report, 1919.

A. Breinl and H. Priestley: "Malaria Contracted in New Guinea by Members of the Expeditionary Force and Its Treatment," THE MEDICAL JOURNAL OF AUSTRALIA, January 29, 1916, page 91.

A. Breinl and F. H. Taylor: "A Malarial Survey of the Township of Cairns," THE MEDICAL JOURNAL OF AUSTRALIA, August 10, 1918, page 109.

A. G. Butler: "Disease of Man Spread by Insects," *Australasian Medical Gazette*, February 24, 1912, page 195.

N. B. Charlton: "Hookworm Disease in the Western Part of Northern Queensland and the Gulf Territory with References to Malaria and Filaria," Report of the Australian Hookworm Campaign Survey, 1922, Number 22, page 2.

R. W. Cilento: "Malaria, with Especial Reference to Australia and Its Dependencies."

J. B. Cleland: "Malaria in New South Wales," THE MEDICAL JOURNAL OF AUSTRALIA, April 3, 1915, page 316.

J. B. Cleland: "Contributions to the History of Disease in Man in Australia," Report of the Director-General of Public Health, New South Wales, 1912, page 228.

Dempster: "Calcutta Medical Transactions," 1835.

R. Dick: "Mosquitoes and Disease," *Australasian Medical Gazette*, January 15, 1912, page 27.

W. J. Doherty: "Fragments of North Queensland History," *Monthly Magazine*, 1929, page 25.

F. Goldsmith: "The Necessity for the Study of Tropical Medicine in Australia," *Transactions of the Sixth Session of the Intercolonial Medical Congress of Australasia*, 1902, page 178.

F. Goldsmith: "Tropical Disease in Northern Australia," *Transactions of Intercolonial Medicine Congress of Australasia*, 1899, page 106.

J. Hudson: "Malarial Fever in New Zealand," *Australasian Medical Gazette*, June 22, 1897, page 267, and May 1, 1898, page 225.

L. H. Hughes: "Some Experiences in the Commoner Tropical Diseases in (late) German New Guinea," The Medical Journal of Australia, January 21, 1920, page 97.

T. H. Johnston: "Notes on Australian Entozoa No. 1," *Records of the Australian Museum*, 1908-1910, Volume VII, page 337.

MacKinley: "Tracks of MacKinley Across Australia," page 387 (cited by Cleland).(7)

Martin (in discussion): "Colonial Fever," *Australian Medical Journal*, 1869, page 178.

R. A. O'Brien: "Anæmia in Ankylostomiasis and Malaria," *Transactions of the Eighth Session of the Australasian Medical Congress*, 1908, Volume I, page 241.

Richardson: *Edinburgh Medical Journal*, December, 1867, page 525 (cited by Hirsch).(6)

A. G. Salter: "Mosquitoes and Disease," *Australasian Medical Gazette*, February 24, 1912, page 195.

P. M. Wood: "Beriberi as seen in the Northern Territory of Australia," *Transactions of the Second Session of the Intercolonial Medical Congress of Australasia*, 1889, page 55.

ON THE NATURE, THE ORIGIN AND THE SPREAD OF CANCER AND TUMOUR GROWTH, WITH SPECIAL REFERENCE TO BIOLOGICAL AND BIOCHEMICAL INFLUENCES AND EXCITANTS AND TO PRECANCEROUS REACTIONS.[1]

By D. A. Welsh, M.D., F.R.C.P. (Edinburgh),
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Of cancer and tumour growth it is difficult to say anything new. Yet there are many facts in the life history of cancer which are not generally known or, if they are known, then their bearing on the problem of cancer is not fully appreciated. I have long been interested in the structural varieties and in the scientific classification of tumours. But much more have I been interested in the life history and biology of tumours, chiefly in regard to (i) their relative malignancy and different invasive capacities, (ii) their genesis and mode of origin and (iii) their growth and mode of spread. I ask you to note, as my discussion developes, how the biology and life history of neoplasia transcend in importance all morphological and histological detail, though, of course, the latter plays an important part in the diagnosis and classification of tumours. I ask you also to keep in mind the distinction between the origin and the spread of a cancer, for these two processes are not necessarily related.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 29, 1929.

On the Nature of Cancer and Tumour Growth.

What is this process that we know as tumour growth or newgrowth or neoplasia? It is one of the most difficult things in the world to define, to say when overgrowth or hyperplasia of tissue becomes newgrowth or neoplasia, to say just at what point does this unique transformation occur, for it is a unique development. It is quite easy with a little training to recognize most forms of newgrowth when they are well developed. But most newgrowths have not sprung into being fully armed like Minerva from the head of Jupiter. They do not rank with the immortals. They have to be born, they have to grow and they may die. Through the mists of ignorance we can at least see that there is a stage in the development of newgrowth when it is neither normal tissue nor yet newgrowth, of a cancer when it is not yet a cancer. That conception of a precancerous stage is fundamental to a proper understanding of the nature of cancer. The precancerous reactions of tissue cells are worthy of the closest study, for they have an important bearing on possible means of preventing the origin and the spread of cancer.

We often say that one of the distinctive characters of tumour growth (neoplasia) is that its development is independent of the needs of the organism in which it takes origin and in which it grows. But I put this to you that neoplasia may be the expression or fulfilment of a need of the parent organism; so that, if we knew more about that need and could do something to satisfy it, then we might be able to avert the onset of cancer. There is some evidence in support of that more hopeful outlook. Again we often say that another characteristic of cancer and tumour growth is its purposeless development, but there too perhaps we take too much for granted; its object may be compensatory or defensive, though perverted and disastrous in its results.

In tumour growth (neoplasia) we meet a new growth of tissue arising in response to an unknown stimulus, developing for an unknown purpose and directed towards an unknown end. Nevertheless, we can often recognize two phases in the life history of a tumour: (i) the development of a neoplasm, that is the transition from normal to neoplastic tissue and (ii) the development of malignancy, that is the transition from non-cancerous to cancerous tissue, from non-invasive to invasive growth.

Now, both the rate at which those developments occur and the stage to which they reach, vary immensely in different tumours, so much so that it is not too much to say that all rates and all stages of neoplastic and of malignant development are represented among the newgrowths that are well known to pathologists. This conception of the progressive development of neoplastic tissue from normal tissue and of malignant growth from non-malignant growth is also fundamental. It recognizes the principle of evolution as applied to tumour growth. And of course this process of evolution and development may be encountered or may be

ILLUSTRATIONS TO THE ARTICLE BY PROFESSOR D. A. WELSH.

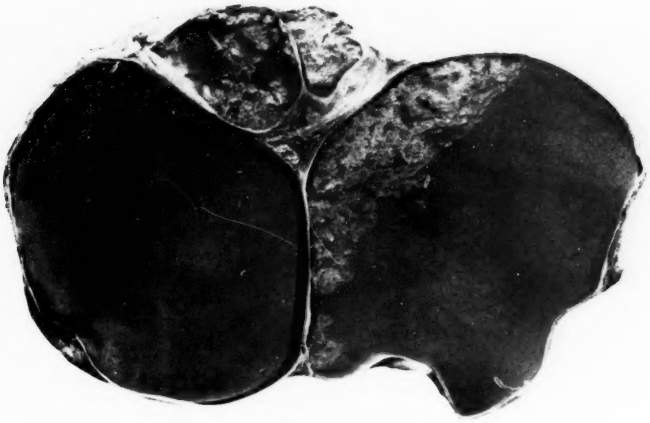
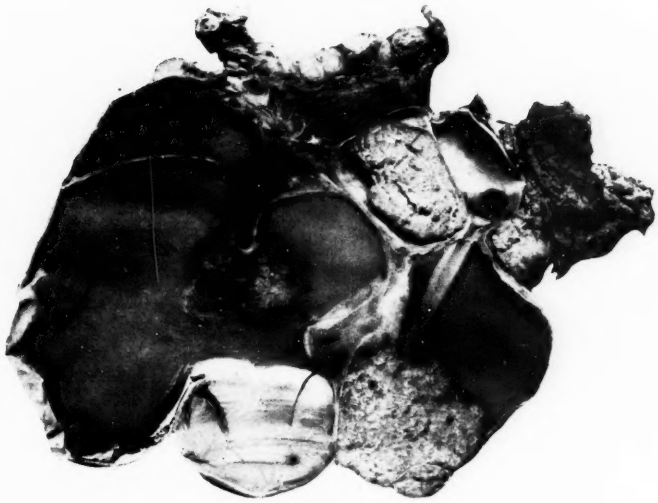


FIGURE I.



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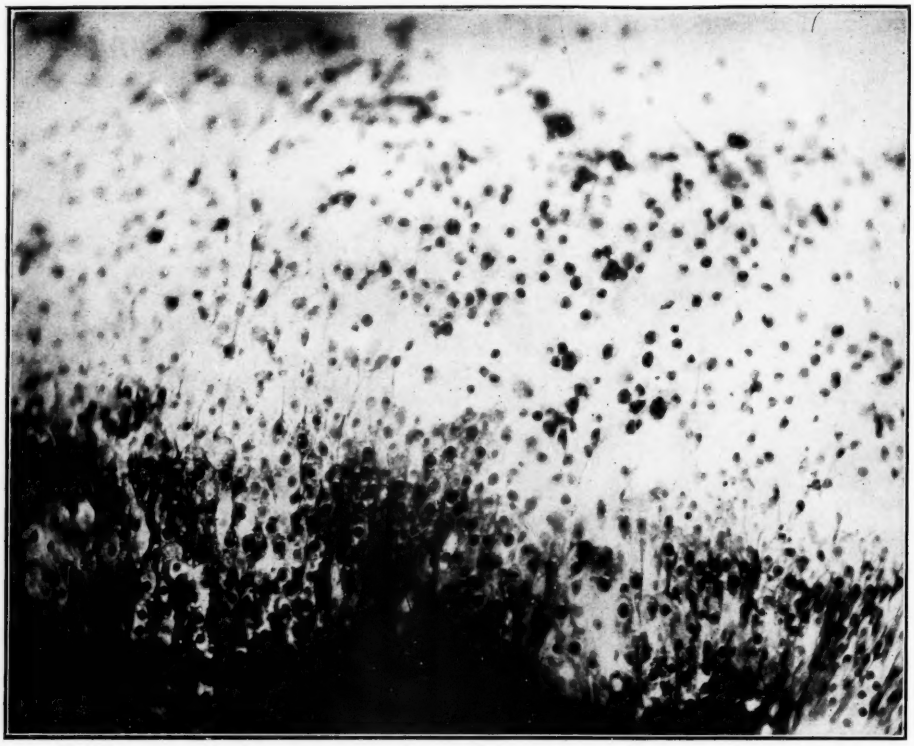

FIGURE I.

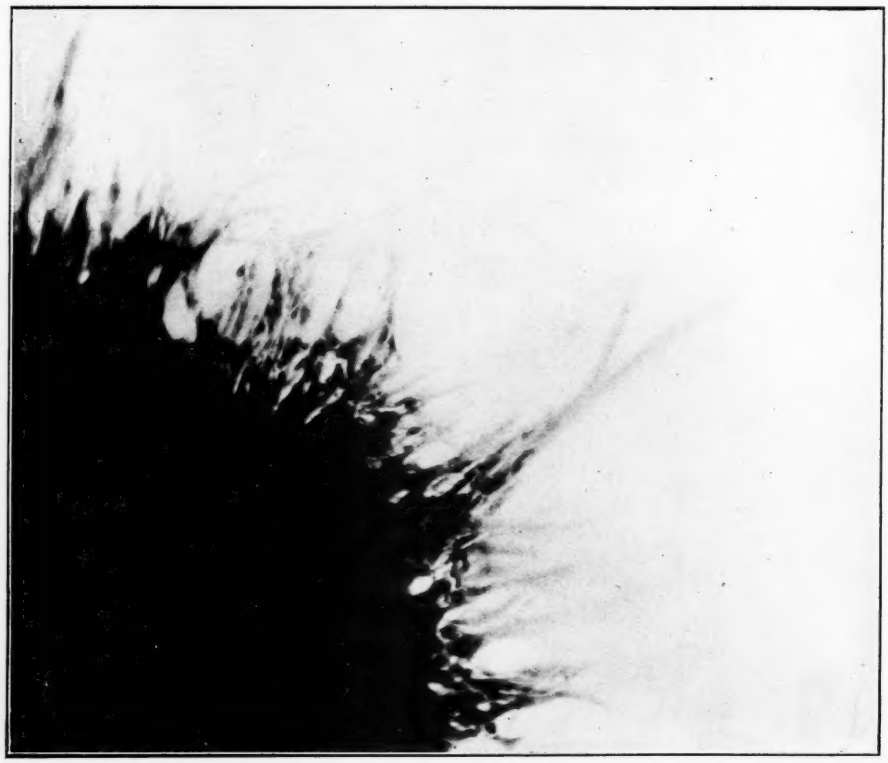

FIGURE II.

arrested at any stage. Hence we meet those precancerous developments of great significance. Hence we find tumours of active growth and tumours of sluggish growth and tumours of quiescent growth. Hence also we have tumours of zero malignancy, of low-grade malignancy, of high-grade malignancy.

Thus we reach that all-important conception of graded or relative malignancy, whereby we recognize that all forms of newgrowth are truly invasive, but that their capacity for invasion varies enormously from a minimum (zero) through all intermediate degrees to a maximum of most intense malignancy. Any idea that there is a hard and fast line between innocent and malignant newgrowths is altogether mistaken and misleading. To put it in another way, all papillomata are not equally innocent and neither are all carcinomata equally malignant. Some papillomata are born with strong invasive tendencies from the beginning, that is they are definitely malignant, as in the case of the papilloma of the trophoblast (hydatid mole); whereas other papillomata may manifest a slowly progressive malignancy and develope invasive tendencies at a later stage in their life history or not at all. So for carcinomata some maintain a remarkably low-grade malignancy, as in the case of the basal cell epithelioma and some squamous epitheliomata of mature acanthoid type; whereas most other cancers are of notoriously high-grade, though noticeably variable malignancy.

The surgeon of the future will ask the pathologist of the future, not the question: "Is this newgrowth innocent or malignant?" but the question: "How malignant is this newgrowth?" Indeed, that is what most surgeons ask now, that one should try to estimate the degree of malignancy of each newgrowth and not merely attach a label like "squamous epithelioma" or "osteogenic sarcoma" and leave it at that. On the whole, our pathological diagnosis is good. It is the cooperation between surgeon and pathologist that is most defective; so that the absence of a good follow up system is one of the most serious handicaps to a better understanding of tumour growth.

On the Origin and Genesis of Cancer and Tumour Growth.

If we cannot yet tell why cancer begins to grow in an epithelial structure, at least we can often tell how cancer begins, how it developes and how it spreads. By a careful study of such facts in its life history we may hope to see the direction in which research into the cause and cure of cancer should proceed. Already we know some of the predisposing causes of cancer and tumour growth in local irritation and trauma, in the influence of heredity and senescence, in the developmental inclusions and embryonic residues of Cohnheim. But that does not exhaust the possibilities, for I hope to show that biological and biochemical excitants play an important part in the origination of most cancers.

A mistake that is often made in discussing these causes of cancer is to treat them as if they were alternative possibilities in all forms of cancer and tumour growth. Because local irritation is known to be the frequent antecedent of many forms of tumour growth, an attempt is made to explain all tumours in that way. Because there is evidence that the origin of many tumours can be traced to embryonic residues, another school of thought attempts to explain all tumour growth on that hypothesis and so on. The truth appears to be that each of the so-called causes, including also heredity, sensescence, biological and biochemical influences, may account in part at least for some cancers and other tumours, but that no single cause is yet known to be definitely related to every form of tumour growth. As a matter of fact, we find that there is often considerable overlapping, so that different combinations of causes may be concerned in the origination of different tumours.

On the Influence of Maturity and Immaturity of the Tissue of Origin.

One of the first things to understand, in order to clarify our minds on this matter, is that many tumours, but few cancers, take origin from embryonic tissues that have been overlooked or displaced in the development of the organism. For a time such embryonic residues cease to develope. Then at a later stage, either before birth or after birth or early or late in life, they may start growing slowly or rapidly, with or without histogenic development, with low-grade or with high-grade malignancy. Thus they give rise to many important forms of newgrowth, some of which are characterized by their undifferentiated or partially differentiated embryonic structure, as in the blastocytoma and the gliomata, while others attain comparative maturity, as in the multiple fibro-myomata of the uterus and the multiple fibro-adenomata of the breast. Heredity may play an important part, both in determining the development of such embryological faults and in determining the predisposition of newgrowths to arise from them. But in any circumstances this cause cannot account for more than a small proportion of the great cancers of adult life.

The majority of the great cancers and of tumours in general take origin from tissues that have reached maturity and have already taken their place in the commonwealth of the fully developed organism. Then something happens to some area of mature tissue, whereby it is induced to assume the characters of a newgrowth and sometimes those of a cancer. What that something is we do not know, though local irritation, trauma, sensescence of tissue and heredity may all be individually or collectively concerned in different degrees in different instances. It is widely accepted that local irritation is the most common antecedent of cancer. But that relation of cause and effect applies more to the cancers derived from the squamous epithelia and less to the glandular cancers. It is further recognized that there is some ultimate and so far unknown cause behind and beyond all the predisposing

causes that I have mentioned. In this unknown cause lies hidden the secret of the origin of cancer and tumour growth.

In my own mind I think of cancer and tumour growth as being mainly due to some biological impulse or some biochemical stimulus, which is sometimes initiated and sometimes precipitated by the predisposing causes above noted, but which sometimes acts independently of any or all of them. I agree that there is probably some unifying and ultimate cause common to all forms of newgrowth, but I recognize that there are many accessory and predisposing factors which may be variously combined in the many different varieties of newgrowth.

On the Operation of Specifically Linked Causes.

Leading up to the operation of biological and biochemical influences in the causation of cancer, I must still further clear the ground. It is a well known fact that in the skin and in the alimentary tract multiple primary origins of cancer are not uncommon. Moreover, those multiple cancers are associated with and preceded by multiple primary foci of precancerous reactions. There can be little doubt that in the skin those phenomena exemplify the influence of irritation as a predisposing cause. In the alimentary tract I am not so sure; for in the intestine this condition of multiple primary carcinomatosis is frequently associated with a condition of multiple papillomatosis, where each separate papilloma gradually evolves a separate carcinoma and there is just as much evidence for this evolution being biological as there is for its being irritational. But in the skin, as Dr. Molesworth has shown,[1] the continued operation of an irritant, like the strong Australian sunlight, over a wide area of exposed skin, like the face and the backs of the hands, may give rise to multiple primary origins of precancerous hyperkeratosis, each of which may develope into a separate epithelioma usually of the basal cell or rodent type.

But even in the skin it is more than probable that a biological predisposition is also concerned, making the development of those cancers and precancers easy for the irritant. For, as Dr. Molesworth again has shown, it is the fair haired person with lightly pigmented skin who suffers most from sun exposure. Heredity is probably not concerned, except in so far as the lessened manufacture of tissue pigments may be inherited as a biochemical predisposition. The incidence of such multiple primary origins of cutaneous cancer illustrates how certain causes may be so closely interwoven as to become interdependent. There is a specific irritant in the form of sunlight; a biochemical laboratory within the body with a reduced output of pigment, creating a specific susceptibility to that irritant and the inheritance of that biochemical laboratory without necessarily any further general predisposition towards cancer. When we note that it is elderly outdoor workers who are most liable to sun-cancer, we have added the last link to a remarkable chain of causes.

On the Operation of Biological and Biochemical Causes.

I ask you to put aside this question of the operation of a local irritant and to let us see if there are not many cancers which are not likely to be influenced in their origin by any form of local irritation. I think we shall find non-irritational origins chiefly among the glandular cancers, for example among those of the prostate, ovary, endometrium and breast, where biological influences and biochemical stimuli dominate those tissue reactions which, passing through precancerous stages, may terminate in cancer.

The Prostate.

In regard to the prostate I used to think that the examination of prostatic enlargements was most uninteresting work; but, after examining a good many prostates for Sir Alexander MacCormick and for Dr. Silverton, I began to find them particularly complicated. Some difficult problems soon presented themselves.

In the first place, the question arose: "Is the primary enlargement of the prostate a truly neoplastic development or merely some form of compensatory hyperplasia?" I think that is an extremely difficult question. Although I have not seen the original paper, I understand that I am not the first to make the suggestion that primary prostatic enlargement, so common in the later years of life, is due to some compensatory hormonal adjustment rendered necessary by the commencing failure of other secreting glands in the body and probably therefore related to some failure of the endocrine system exhausted with advancing years.

Then there arose the further question: "How does cancer begin in the prostate?" When I first began examining enlarged prostates, I found that most of them were wholly simple, undergoing that adenomatous overgrowth which is now recognized to be some form of compensatory adjustment, whereas a few were wholly cancerous. Since it was my practice to take microscopic sections from each lateral lobe and from the middle lobe, that was rather a remarkable finding. It was strange that an enlarged prostate should throughout its whole area undergo so generalized a cancerous development that no trace of non-cancerous prostatic epithelium was left. It was, of course, impossible to assume that so complete a cancerous transformation could have taken place without some progressive evolution from non-cancer to cancer.

It did not take long to find the solution. I began to notice foci of true cancerous development, along with other foci of precancerous development, scattered throughout the substance of the prostate, which was already enlarged by compensatory overgrowth. These multiple separate foci of cancer and of precancer were found everywhere throughout the enlarged gland, often deeply placed in its substance and, if they were recognized in one lateral lobe, they were sure to be found in the other also. At first they were so widely separated one from another that it was impossible to infer that the develop-

ment of one cancer area had influenced the development of another. It was obvious that the cancer cells were taking origin *in situ* from the prostatic glandular epithelium already stimulated to simple adenomatous overgrowth. Hence the conclusion was inevitable that this multifocal origin of cancer throughout an already enlarged prostate was due to the fact that the whole prostatic epithelium was reacting similarly and more or less simultaneously to a common biochemical stimulus. That stimulus had induced the whole prostatic epithelium to undergo a compensatory overgrowth; it had then succeeded in inducing multiple separate areas of that epithelium to undergo a cancerous transformation and, given time, it would doubtless succeed in transforming every prostatic epithelial cell into a cancer cell. Irritational influences can be definitely excluded, as the evidence of prostatitis is negligible in such cases, but heredity may be concerned in so far as a predisposing type of endocrine system may be inherited, that is again heredity may exert an influence through the biochemical laboratories of the body.

The Ovary.

As regards the ovary, the origin of cancer there is interesting and very similar to its origin in the prostate. In my experience most cancers of the ovary are primary and most have been preceded by a more simple form of newgrowth, usually a papilliferous cystadenoma, traces of which can be detected even when the cancerous transformation has reached an advanced stage. In many instances both ovaries are involved and both are similarly involved, though not always to the same extent. Bilateral developments in one ovary may lag a little behind those in the other, but not very far. As in the prostate, these facts point strongly to some biological influence or biochemical stimulus operating on both ovaries, just because they are ovaries and are therefore exposed to and react to the same excitants more or less simultaneously. I think a strong case can be made out in support of that hypothesis. The whole epithelial area in the substance of both ovaries, under the influence of some common excitant, undergoes a preliminary neoplastic development, which is not usually in the first instance carcinomatous, but in which cancer tends later to develope at separate primary foci and these foci may ultimately coalesce. Hence, as in the prostate, we again get a multifocal origin of primary carcinoma which cannot be explained by any form of local irritation, but must be referred to biological and biochemical causes operating on the ovaries as such.

In support of this hypothesis, I show you a remarkable specimen, presented by Dr. F. A. Maguire, where both ovaries have been transformed into papilliferous cystadenomata, within which is proceeding the development of bilateral and multifocal carcinoma. The specimen is remarkable because the multiple separate foci of cancer are all at an early stage of development and because they can be seen to take origin within the papilliferous cysts of both ovaries. It is impossible to imagine that such a carcinoma was introduced to the ovaries from without. Until this key specimen was presented, I had only indications of the multifocal origin of primary cancer of the ovaries, but with this specimen I have proof. My point is that in this specimen, illustrating the early stages, you can actually see primary multifocal carcinoma beginning within the papilliferous cysts of both ovaries and again that in the specimens of more advanced ovarian carcinomata, which I also show, you can still trace the remnants of the papilliferous cystadenoma which preceded the carcinoma.

Now I do not say that that is the only way in which primary carcinoma of the ovary developes, but it is certainly one way and it happens to be the principal way represented in our museum collection. That collection goes back over many years and the specimens were not selected by me, but were presented at different times by many different donors. So I take it that we have a representative collection.

Bland Sutton has had an experience of ovarian carcinoma just the reverse of mine.[2] He finds that primary cancer of the ovary is rare, but that secondary cancer is common, especially when the primary growth has developed in the breast or in the gastro-intestinal tract or in the gall bladder. He offers an explanation almost in those words. Arising in the mucous membrane of the stomach, the gall bladder or the colon, the cancer slowly permeates it and implicates the submucous, muscular and peritoneal coats. The cancer cells can then escape freely into the great serous cavity and be distributed by the fluid, aided by the movements of the bowels and gradually reach the pelvis. In the pelvis the most obvious organs on which they could fall, would be the ovaries, as they so often rest on its floor. In such conditions the ovaries receive a covering of falling cancer cells, as shrubs are clothed by snowflakes in winter.

All that I can say is that, among the many hundreds of abdominal cancers that I have seen, I have never met such a condition as is pictured by Bland Sutton, that is where the ovaries are secondarily infected by cancer cells to such an extent as to give rise to the clinical signs of ovarian carcinoma and yet when the abdomen is opened up, there is no generalized infection of the peritoneum such as would indicate the presence of a primary cancer higher up. I do not mean to imply that such a condition never occurs. I accept the statement that other observers have seen it, though I have not. All my experience, however, leads to the conclusion that Bland Sutton has not described the most common way in which the ovaries develope carcinoma, but that the hypothesis that I have submitted and the specimens that I have demonstrated, illustrate a different and much more common origin.

The Breast.

In regard to the breast, perhaps the origin of carcinoma there is not quite parallel to its origin in the prostate and in the ovary. In the breast it is probable that local irritation plays a more im-

portant part, though not such a dominant part as is generally supposed, for here again biological and biochemical causes are strongly represented. In the breast, too, the multifocal origin of cancer is not so obvious, but even in the breast the progress of a cancer is represented not altogether by an invasive spread of cancer from a single lobule, but also by the diffusion of a carcinomatous change from lobule to adjacent lobule. The lobules that are going to become cancerous *in situ*, develope a precancerous state of unrest and excitement among their epithelial cells, which react in many different ways, until a definitely carcinomatous stage is reached. At all events cancer of the breast does not originate from a single lobule of breast tissue.

What happens is this, that within the cancer area lobule after lobule becomes involved first in a precancerous reaction and then in a cancerous process and quite often this progressive involvement of successive lobules can be traced under the microscope, as I shall show you. Of course, side by side with this evolution of the cancer process, which is equivalent to a process of multifocal origin in which the foci are closely grouped instead of being widely separated as in the prostate and in the ovary, a cancerous invasion of the breast tissue is going on from the older cancerous lobules; but up to a certain stage the separate development of each process can be traced.

Ultimately in advanced conditions all those distinctions are lost, as one would expect, and the whole cancer area becomes so crowded with invasive cancer cell masses that the recognition of separate lobular areas of cancer becomes impossible.

I have made a special study of the precancerous reactions of mammary tissue and I find that biochemical (hormonal) excitants are much more often indicated than any irritational cause. It would take too long to give the evidence now; but my general conclusion is that true mastitis (in the sense of a definite inflammatory reaction) is not the common cause of mammary cancer, but that that cause must be sought, as in the prostate, in some biochemical stimulus or hormonal adjustment to which breast tissue is peculiarly sensitive.

The Uterus.

In regard to the endometrium, there is evidence that the development of the common adenocarcinoma is related more to biological and biochemical causes than to any form of local irritation. But, as I have discussed this elsewhere,[3] I need only mention it here, though I shall show you illustrative specimens.

Cancers of the *cervix uteri* are much more often squamous epitheliomata, which can frequently be traced to some form of irritational origin, as is usual in that type of cancer.

On the Growth and Spread of Cancer.

In regard to the growth and spread of an established cancer many interesting points arise. But what I want to say has reference chiefly to the local spread and extension of the original cancer area. In all probability two fundamentally different processes are involved.

Invasive Growth.

In the first place there is the process of invasive growth, with which all are familiar, including (*a*) the growth of the cancer mass by division and multiplication of the cancer cells already formed and (*b*) the invasion of adjacent tissues by the multiplying cancer cells. Undoubtedly this combined process does contribute materially to the local extension of the cancer area. On an epithelial surface, for example, the invading cancer cells may go on undermining the marginal epithelium and so lead to the extension of a malignant ulcer. In a gland like the breast the mass of a scirrhous cancer is continually being increased by an invasive growth of the cancer cells. And, of course, the permeation of lymphatics and the development of more distant metastatic growths are but extensions of this invasive process by the actual carriage and transplantation of the cancer cells.

Contact Spread.

But there is another process, that of contact spread, with which you are probably not so familiar, and yet it is one of the most important means by which the immediate local extension of an established cancer is brought about. It consists in the passage of a malignant influence from cancer cells to adjacent non-cancerous epithelial cells, whereby the latter are induced to become cancerous *in situ*. I have seen this now in hundreds of specimens in which the contact zone has been examined, that is in which a cancer makes contact with its marginal epithelium. One sees it in the skin, in all mucous membranes lined by squamous epithelium, in the alimentary tract, in the endometrium. I am satisfied that it is the chief means by which untreated cancer spreads to involve a surface epithelium; and, even in the glandular cancers, like those of the breast, there is evidence that a similar contact spread of cancer obtains, in addition to the invasive spread. This local transmission of a cancer impulse from cell to cell is probably the explanation of such developments as Paget's disease of the nipple and breast, for that low-grade type of carcinoma simply follows the lines of contact along a closed surface, outwards from mammary ducts to skin and inwards along the ducts to the gland acini.

When a definite focus of cancer has emerged from its precancerous state, it begins to grow and spread, but the causes of its growth and spread are not necessarily the same as the causes of its origin. In fact, we know that a new set of conditions begins to operate when a cancer is established. Those new conditions do not exclude the continued operation of the original cause of the cancer, but they are additional to and often more powerful than the cause of origin in promoting the subsequent growth and spread of the cancer. In other words, a cancer is created by the cooperation of certain causes, known and unknown; but the cancer itself creates certain conditions which did not exist before the

cancer appeared, and those conditions promote the further growth and spread of the cancer. Hence the greatest stimulus to cancer growth is cancer growth.

What that stimulus is, we do not know. It might be an ultramicroscopic microbe, the true cancer parasite, but there is no convincing evidence in support of that hypothesis. It is more probable that the stimulus is analogous to that growth-promoting substance which can be extracted from embryonic tissue and added to a tissue culture to excite further growth, and which is found to have multiplied itself in the process of exciting tissue growth. But the stimulus with which we are now concerned, is not an ordinary growth-promoting stimulus, but an extraordinary cancer-promoting stimulus. It does not merely stimulate an epithelial cell to renewed multiplication and growth. It completely transfigures a somatic epithelial cell, as it lies in the tissues, causing it to pass through definite and recognizable precancerous reactions which terminate in the maturation of a fully developed cancer cell. That cancer-promoting stimulus has therefore succeeded in imparting to an adjacent epithelial tissue cell *in situ* all the unique characters of a cancer cell, including (i) the power of independent growth, (ii) the capacity for invasion and (iii) the ability to pass on to other epithelial tissue cells that cancer-promoting stimulus which it had received.

The local contact spread of an established cancer may be continuous or it may be discontinuous. The continuous contact spread is more easy to understand, but that the local dissemination of a cancer-promoting stimulus may be discontinuous, is also certain. The contagium, whatever it may be, may proceed *per saltum*. I have seen it thus proceeding in the larynx and in the lip, specimens of which I show, where the presence of one primary focus of cancer growth excites the development of another primary cancer adjacent to it. So a neglected cancer of the lower lip may spread by the stimulation of successive crops of adjacent primary foci, until the whole lip is riddled with them. It is reasonable to suppose that, if the original cancer had been excised, the other foci would never have developed. But time does not permit me to pursue this fascinating subject.

On the Development of Multiple Primary Foci of Cancer.

So far my discussion of the origin and spread of cancer has disclosed three different ways in which multiple primary foci of cancer may develope separately but not unrelated.

The first way is illustrated by the growth of multiple separate epitheliomata, chiefly of the basal cell or rodent type, on areas of skin exposed to strong sunlight. That is an example of a cancerous process in which the continued operation of the original irritant over a long period of time gives rise to multiple primary foci of similar, but not always identical, growth at different times and at different places. The origin of one focus of the cancer is independent of the origin of any other focus, except in so far as they are related through the operation of a common irritant (sunlight) acting on a common tissue (skin) having a common biochemical predisposition (lack of pigment) not necessarily to cancer, but certainly to the specific irritant, while age, as ever, adds its quota to the general causation.

The second way is illustrated by the multifocal origin of cancer throughout a gland like the prostate, where, owing to certain hormonal adjustments rendered necessary in the later years of life, the whole gland undergoes a compensatory adenomatous overgrowth which may be succeeded by a carcinomatous development of a uniform type, occurring more or less simultaneously at separate foci throughout the gland substance and ultimately overtaking every epithelial cell in the gland within a short period of time. That is an example of a general biochemical stimulus operating on the whole epithelial content of a gland, because that gland acts as a single physiological unit, all of whose cells probably work in specific relation to the endocrine system.

The third way is illustrated by the discontinuous dissemination of the cancer-promoting substance, as above described in the case of the lower lip.

Legends to Illustrations.

Figures I and II.

Figure I and Figure II illustrate a primary papilliferous cyst-adenoma, developing into carcinoma, of the right and left ovaries from a woman thirty-eight years of age. For the last six months there was a history of irregular and profuse hæmorrhage, increasing at menstrual periods, but without pain. Examination revealed two large irregular masses in the pelvis and the clinical diagnosis lay between uterine fibroids and bilateral cancer of the ovaries (on account of the size and rapid growth of the masses).

In Figure I the epithelial newgrowth is still confined within the capsule of the ovary. In Figure II the epithelial cell masses have burst through the capsule and are ready to infect the peritoneum. There is little doubt that this represents a primary bilateral multifocal carcinoma in the making and it clearly shows the progressive and independent multiple origins of the growth in each ovary; not one ovary infected by the other (although one is more advanced than the other) nor both infected from the alimentary tract. That interpretation was confirmed by the microscope (Dr. F. A. Maguire's case, Museum, page 1357).

Figure III.

Figure III illustrates a more advanced stage of primary carcinoma of the ovary developing from a papilliferous cyst-adenoma, traces of which can still be seen. The cancerous growths have become confluent. The peritoneum was studded with secondary growths. The patient was fifty-three years old (Museum, 1236).

In each figure a complete hemisection of the ovary is shown, reduced approximately to five-sixths of its natural size.

References.

(1) E. H. Molesworth: "Rodent Ulcer," The Medical Journal of Australia, June 18, 1927, page 878.

(2) J. Bland Sutton and A. E. Giles: "The Diseases of Women," Seventh Edition, London, 1916, page 351.

(3) D. A. Welsh: "Cancer of the Uterus: Some Facts in its Life History and their Clinical Significance," *The Journal of the College of Surgeons of Australasia*, November, 1929, page 254.

MINOR DISTURBANCES OF NUTRITION IN CHILDREN.[1]

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CLINICAL experience leads to the belief that while the signs and symptoms of established disease in children are generally well recognized and adequately investigated, the minor manifestations of petty disturbances of health that are such an annoyance to the parents and are so often passed over by the doctor with a bottle of medicine and only temporary relief, do not receive the attention they deserve. We are too apt to think of the sick or ailing child in terms of the text book classification of diseases. Let us turn first to the healthy child and get some conception of what is the normal state, not only of absence of complaint, but the normal physical and mental condition, the appearance, height, weight, skeletal and muscular growth, state of the skin, hair and teeth, the subcutaneous tissues, the blood, the lymphatic system, the development of the sexual organs, the mental development and the reaction of the nervous system. Then and then only can we appreciate the minor deviations from health and prevent or at least mitigate the ravages of disease.

It is absurd to continue to regard, as we have done in the past, the measurements of weight or of height and weight as adequate criteria of normal growth. Yet almost every definition of malnutrition is based on deviations from an average height-weight ratio.

It must be realized at the outset that so many influences affect the growth of a child that it is impossible to attribute any condition of malnutrition to any single cause; but with due regard to hereditary and congenital factors and environmental conditions, the diet must always remain the basic factor in nutrition. In this country we see relatively little actual starvation and the conditions due to specific vitamin deficiencies that come under our notice, are comparatively ill-defined and mild. The human infant, too, shows an extraordinary adaptability to variations both in quality and quantity of foodstuffs; yet it is not unimportant that children should be supplied with foods that are sufficient in quantity and physiologically best for growth and development. I cannot review in detail the physiological requirements of the growing child, but I may be allowed to outline briefly a few points that I consider specially important.

Protein requirement has been a matter of controversy for many years and there is still no general agreement on the subject. It has been argued that because the protein of human milk is diminished in the later months of lactation, the child therefore requires less protein; but Nature surely never intended that once a child has developed teeth with which to chew, it should still be kept on a liquid diet until the supply is exhausted. As long as the breast milk is sufficient, the baby will thrive because the biological value of the protein is adequate for growth, but when cow's milk or other artificial foods are used, it is quite otherwise and it may be necessary to double the amount of protein that would be adequate with human milk; for it is a matter of common knowledge that proteins have biological values according to the amino-acids they contain and that when dealing with artificial feeding, it is the mixture of proteins rather than the total amount of protein that is important. Thus animal proteins may be said to have about three times the value of vegetable proteins. I am therefore in agreement with Holt who states that in a mixed diet 15% of the total calories should be supplied by the protein; at least one third of that should be of animal origin.

It was thought at one time that carbohydrates could entirely replace the fat in a diet without detriment to the health of a child; it is now recognized that fat is essential and that fats also vary greatly in biological value according to their content of the fat-soluble vitamin *A* and the closely associated antirhachitic vitamin *D*. If protein, fat and carbohydrate are properly balanced, there is never any difficulty in the assimilation of fat; this has a direct bearing on the so-called fat intolerance which occurs almost entirely where the tolerance has been lowered by long continued deficiency of fat in the diet.

Carbohydrates supply some 50% of the calories in a mixed diet and it is of some importance that the starches should not too far overbalance the pure sugars; so-called carbohydrate dyspepsias are more often starch dyspepsias.

The adequate supply in balanced quantity of the various vitamins and the balance of mineral salts are matters of prime importance in any dietary, but in practice, provided the food supply is abundant and varied, they need not cause any concern.

What, then, are we to regard as indications of malnutrition? In the baby restlessness, fretfulness and capricious appetite may be present with perfectly normal gain in weight and apparently normal development and *vice versa* a perfectly contented, happy baby may fail to gain or develope at the normal rate and again a child with no symptoms and normal gain in weight may yet exhibit definite signs of malnutrition. It must be remembered that it takes many months of faulty feeding to produce gross defects in growth and development and that, therefore, the defects we look for can be of only minor degree.

In the baby, therefore, I am accustomed to note the presence or absence of fat, the firmness or otherwise of the skin and underlying tissues and muscles, the colour and pigmentation of the skin, the visibility of the subcutaneous venules, a bluish tinge of the sclerotic membranes, abdominal fullness or distension, the beginnings of a Harrison's groove or of a rosary, the character of the stools and head sweats as well as delay in teething, sitting, walking

[1] Being a lecture delivered under the auspices of the Melbourne Committee for Post-Graduate Work, in August, 1929.

and talking. The colour of the sclerotics is in most cases the earliest warning noted.

In older children the indications of early malnutrition that are noted, are legion: Failure to gain or loss of weight, loss of appetite or perverted appetite, lassitude, attacks of syncope, "bilious" attacks, constipation, persistent pains in the abdomen, headaches, restlessness at night, night terrors, enuresis, tics, stuttering, nervous manifestations of all kinds, even anomalies of conduct, sweats, tendency to frequent attacks of bronchitis, asthma, recurrent sore throats, rheumatic pains, undue tendency to infections of all kinds and poor resistance to such, as also failure at school, lack of ambition whether at work or at play, and habits of aloofness.

On examination of these children we may find many or few defects, bony defects, poor musculature, disproportion between skeletal and muscular growth which is responsible often for the milder types of scoliosis, disproportionate growth of different parts of the body, defective teeth, unhealthy gums, alterations in skin, such as roughness, scaliness or transparency so that the subcutaneous venules are unduly visible, or loss of elasticity or of skin "body," increase in hairy covering ranging from the thin downy hair seen down the spine of ill-nourished children, particularly in association with sweating, to a generalized downy covering or even a coarser type of hirsuties, pallor or sallowness often with vasomotor instability and recurrent albuminuria, a type of blood characterized by retention of the infantile features of lymphocytic preponderance and extreme variability in size of the lymphocytes, enlarged lymphatic glands and general hyperplasia of lymphoid structures as shown by McCarrison to be readily caused experimentally in monkeys; hence the tendency to enlarged tonsils and adenoids, to appendicitis, mucous colitis and so on. It may be noted that a high carbohydrate diet increases the lymphocytes, while a high fat diet increases the polymorphonuclear cells of the blood. In the type of anæmia variously known as "alimentary" or "school" anæmia, it is generally found that there is little or no diminution of red cells, little diminution of hæmoglobin, but a relative lymphocythæmia; this condition seems to be most constant in those children who are continually complaining of vague rheumatic pains.

Acidosis is found mainly in children who are habitually fed on an ill-balanced diet in which starchy foods predominate. The sequence is a diet poor in protein and in fat, with excess of carbohydrates, particularly starches, then intolerance of all fat, anorexia and finally vomiting, abdominal pains and fever. While the immediate effects are mitigated by feeding easily assimilated sugars, a continuance of high carbohydrate diet leads inevitably to frequent recurrences. The only means of prevention is the adoption of a well balanced diet. Unfortunately there has been of late a tendency to shelter behind the old word or conception "diathesis" as an explanation for many of the conditions I have described. Were the term restricted to the sphere of hereditary and congenital tendencies, I should not quarrel with it, but it has been used in such an all-embracing sense that its use simply clouds any clear thinking attempt to define causes. The nervous child, while the exciting cause of the nervousness may be sought elsewhere, is found most commonly among this group of malnourished children and the behaviour of delinquent children is distinctly improved by generous feeding. The so-called debilitated child is essentially a malnourished child.

The means that should be taken to prevent malnutrition will be realized from what I have already said. Of course, much remains to be said on the subject and further investigations are required, but nevertheless in the past twenty years during which I have interested myself in this subject, all research has tended to confirm and to modify only in minor respects the opinion which I developed and expressed in writing in 1913, that most children get far too little protein, too little fat and a great excess of carbohydrates, especially in the earlier years of life. I am totally opposed to the ideas that have been foisted upon us by past generations that children cannot digest this or that article of food and to the restrictions in diet thereby imposed without an iota of scientific proof of their validity. Cereals are not the most easily digested of foods, eggs are not "bilious," meat does not cause kidney disease or high blood pressure. Put in front of children plenty of mixed fresh vitamin-containing foods and allow them to choose what they want; they are far better judges than their mothers of what they need. The earlier a child is given a mixed diet, the more readily will it take it. It is my custom to give in addition to the breast milk at eight or nine months of age such foods as eggs, meat juice and gravies, fruit and vegetables. I never wean with cereals. Bottle fed babies are put on as strong cow's milk mixtures as they can take and at six or seven months these additional foods are given. In all cases a child at ten or eleven months is having practically an adult diet. Rarely among children fed in this way do I find cases of difficult feeding nor, unless children are made to take what they do not want, does persistent anorexia occur.

ON TETHELIN AS A TISSUE CULTURE MEDIUM.[1]

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THE purpose of this investigation was to observe the effects of "Tethelin" on embryonic epithelium

[1] This article is based on an article published by the same authors in *The Australian Journal of Experimental Biology and Medical Science* of September 16, 1929. This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

when cultivated *in vitro* in a medium of plasma containing a given saline solution of "Tethelin."

The growth rate in this medium was compared with controls made in a medium consisting of plasma and chick embryonic extract.

Early in the history of tissue culture technique it was shown that plasma and serum inhibit the indefinite multiplication of tissues and isolated cells *in vitro* and consequently the growth-promoting substances which must be added to the coagulated plasma in order to enable tissues to proliferate *in vitro* for indefinite periods, have been the subject of a most intensive investigation.

Of these nutritive media, embryonic extract has proved the most successful and apparently one of the most suitable growth-promoting substances; at the same time the trephones, as these growth-stimulating substances are called, appear to be proteins of very unstable nature.

Many authors have attempted to determine the nature of these substances from which cells synthesize new protoplasm, and also to isolate the principle responsible for growth. In view of these recent researches the effect of this growth-stimulating lipoid ("Tethelin") when employed as a tissue culture medium is of special interest.

"Tethelin" is one of the three supposedly pure lipoid secretions of the anterior lobe of the hypophyis and was isolated by Brailsford Robertson. The remaining two substances, cholesterol and lecithin, have been found to be either non-stimulating or actually retarding in their influence on the growth of neoplasms and the animal body, while experiments *in vivo* on the growth-stimulating nature of "Tethelin" have been demonstrated both in normal animals and in rats bearing transplantable neoplasms.

Throughout this investigation the glandular wall of the intestine was removed under sterile conditions from a seven day chick embryo and cut into very small fragments. Plasma obtained from the ventricle of adolescent chickens was employed as a supporting medium.

The following media were then added in equal volumes to the cultures: (i) a solution of one hundred milligrammes of "Tethelin" in twenty-five cubic centimetres of sterile normal saline solution of pH 7·5; (ii) equal volumes of embryonic extract and a solution of one hundred milligrammes of "Tethelin" in saline solution of the same hydrogen ion concentration; (iii) embryonic extract was obtained from seven day chick embryos.

The cultures, after twenty-eight hours' incubation at 38·5° C., were fixed and stained.

An examination of these preparations showed that this growth-stimulating lipoid so greatly increased the initial growth rate that an extensive membrane was formed after twenty-eight hours' growth incubation (see Figure I). This membrane of new growth contrasted most definitely with the small, tongue-like migration of cells observed in the control cultures after a similar period of incubation in embryonic extract (see Figure II).

In summarizing this investigation the authors have succeeded in confirming the growth-promoting qualities of "Tethelin" *in vitro*.

Legends to Illustrations.

Figure I.

Photomicrograph of portion of the periphery of an explant cultivated in plasma and "Tethelin," showing extensive membrane formation after twenty-eight hours' incubation. Observe the healthy condition of the cells.

Figure II.

A portion of the periphery of a control culture taken with a lower magnification. Compare the extent of the proliferation and the absence of membrane formation with those of Figure I.

Reviews.

THE SURGERY OF MINOR CONDITIONS.

"MINOR SURGERY" by Christopher should commend itself to surgeons and general practitioners, for it deals with conditions that are too often only cursorily dealt with in the average text book.[1] But, as the author admits, it is impossible to deal with minor without encroaching upon major surgery. In actual practice it is found very frequently that the minor condition may develope into the major with startling suddenness. It is also found that a considerable portion of the average surgeon's work consists not of the "big things of surgery." Hence to all a knowledge of the "lesser things" is of transcendent importance; indeed, it is through the possession of this knowledge that they will prevent some of the major complications occurring.

The book reveals an intimate acquaintance with modern methods of treatment, such as radium, X rays and diathermy. Incidentally, the author speaks highly of X rays in the treatment of erysipelas. Injuries and infections of the trunk occupy nearly seventy pages; this will give an idea of the wide scope that the book covers. Information is given that would otherwise be obtainable only by reading several text books.

In the chapter on breast tumour the author makes a statement about being guided by the results of a frozen section; with this many surgeons will be inclined to disagree. His quotation from Bloodgood on "The Diagnosis and Treatment of Breast Tumours" is extremely valuable for any surgeon to refer to in that most difficult of all subjects.

A chapter on intracranial injuries suggests a limited personal experience on the part of the author or he would be more insistent on the value of lumbar puncture.

The interesting chapter on foreign bodies in various regions is a very useful contribution in a book of this nature.

In dealing with Raynaud's disease no mention is made of Royle's useful operation of ramisection.

In dealing with gas gangrene which is very far from being a minor surgical condition, the author does not call attention to the fact that the whole length of muscle or muscle groups may be affected, hence high amputation may be necessary.

The chapter on minor surgical technique includes among others such widely varying procedures as local anæsthesia, intubation, skin grafting, blood transfusion and the application of plaster of Paris.

The book is well illustrated and can be recommended with confidence.

[1] "Minor Surgery," by Frederick Christopher, M.D., F.A.C.S., with a foreword by Allen B. Kanavel, M.D., F.A.C.S.; 1929. London: W. B. Saunders & Co. Melbourne: James Little. Royal 8vo., pp. 694, with illustrations. Price: 37s. 6d. net.

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SATURDAY, MARCH 1, 1930.

Patrick Manson.

ZONAE TORRIDAE TUTAMEN! This phrase epitomizes the self-imposed task of the Royal Society of Tropical Medicine and Hygiene of which Patrick Manson was the first president. Thirty years before he, Sir James Cantlie and others eminent in the world of tropical medicine founded this famous society, Manson had demonstrated that the causative agent of a certain tropical disease was conveyed by a particular species of mosquito. The significance of this discovery is now evident. Manson's discovery in Amoy in 1877 was the result of patient study and keen observation carried out in a tropical environment. He found the filarial worm in the blood of patients in the little hospital and in their homes. He traced the worm to the stomach of the mosquito *Culex fatigans* and demonstrated that it passed through its developmental stages in the thoracic muscles of the mosquito, eventually to be discharged into the cutaneous tissue of the human being. Manson thus demonstrated for the first time that a tropical disease is transmitted to man by a mosquito. As a result of this study and discovery Manson sought and found evidence of a similar transmission of the plasmodium of malaria. He carried his investigations a long way toward the goal of actual proof and stood behind Sir Ronald Ross with encouragement and counsel until the latter overcame the last difficulties and revealed the wholy story of malarial infection. Manson's discovery led the way to the elucidation of the problem of trypanosomiasis, yellow fever and dengue fever. No one can challenge the assertion that it was Patrick Manson who founded modern tropical medicine, that his work enabled those who followed him, to gain a mastery over some of the deadliest of the tropical diseases and that the society founded in 1907 was justified in setting itself the task of rendering tropical countries safe for humanity.

The medical profession in Australia has a special interest in the safeguarding or protection of tropical areas. Part of Australia lies within the tropics; Australia is responsible for the proper government and control of large islands in the Pacific; Australia has a special interest in the health conditions of all the islands in the eastern half of the Pacific Ocean. The medical profession in Australia has honoured Peter Bancroft whose great work in connexion with the filariæ has a special bearing on the pioneering investigations of Patrick Manson. In a leading article published in *The Times* of December 18, 1929, it is pointed out that "a world which has done honour to Pasteur and Lister freely and with alacrity, has shown itself indifferent towards Manson." The greatness of Patrick Manson has not been adequately recognized by the members of his own profession. Little wonder that the non-medical world is ignorant of its immense debt to the father of tropical medicine.

Dr. G. Carmichael Low, the President of the Royal Society of Tropical Medicine and Hygiene, Sir Ronald Ross, Sir Andrew Balfour, Dr. J. W. W. Stephens, past Presidents, Dr. Arthur G. Bagshawe, Honorary Treasurer, and Dr. C. M. Wenyon, Honorary Secretary, are making an appeal throughout the British Empire to those who realize how much Manson had done to increase the prosperity of the Empire. It is proposed to perpetuate the name of this great man by erecting a suitable house for the Royal Society of Tropical Medicine and by naming it Manson House. There are at present some fifteen hundred fellows of the society, including the leaders in tropical hygiene of practically every country. The fellows and some of the friends of the society have subscribed the sum of £6,000 toward the fund. It is estimated that £20,000 will be required for a suitable building wherein the work of Manson may be continued and extended. Sentiment plays a large part in the lives of men. Medicine and the welfare of the human race are not matters of cold business, of full value for money spent. The fellows of the Royal Society of Tropical Medicine and Hygiene are appealing outside the

small professional circle of workers in tropical medicine and are relying on sentiment to enable them to erect a monument worthy of the cause. The house will not be merely a structure of architectural artistry, although beauty should not be despised. Suitable premises are urgently needed for those engaged in the work begun by the father of modern tropical medicine, in order that full advantage may be taken of debate, discussion and exchange of ideas. We appeal to the sentiment of the medical practitioner in Australia to add a substantial quota. Contributions may be sent to the Editor of this journal. They will be acknowledged in these columns. It is essential, however, that those who wish to associate themselves with the proposal, will act at once. As a young man Manson worked for work's sake and faced difficulties, dangers and discomfort so that he could contribute something substantial to the store of the world's knowledge. His contribution was larger than he anticipated. But he did not attempt to balance his ledger. Remuneration was of secondary importance. Manson's work has produced a vast economic advantage to the Empire and to the world, since by transforming into healthy districts tropical countries where deadly infections earned for them the name of the white man's grave, the white man has been enabled to exploit the natural resources of the whole tropical zone. It is not this indirect result of Manson's great work that we should honour. It is because Manson gave a promise of life and health to native and white man in the tropics that we owe so much to him. We trust that Australia's contribution to his memorial will be worthy of the size and importance of this outpost of the Empire.

Current Comment.

LYMPH STASIS AND CANCER.

It will be remembered that seven or eight years ago Sampson Handley sought to apply the lymphatic permeation theory of metastasis to cancer in all its forms. His book on the subject was read eagerly by pathologists and surgeons. Though his views may not have been universally adopted, they were accepted in so far as they influenced the radical removal of such organs as the breast for malignant disease. The subject was discussed in these columns on February 24, 1923. As has been stated recently in this journal, Handley has expressed the opinion that lymph stasis plays an important part in the ætiology of malignant disease.[1] He has supported his views with observations and arguments which merit careful consideration.

He begins by considering the origin of many celled organisms. He regards it as likely that they arise from some inhibition of the process of cell division when it has proceeded half way, that is to say, the nucleus undergoes complete division, but the cell protoplasm still remains connected by protoplasmic bridges. He postulates for the primitive one-celled organism an irresistible appetite for food and for multiplication and points out that these are the appetites that dominate the cancer cell. Cancer in his opinion thus becomes an atavistic reversion of certain cells of the body to the state of their primitive one-celled ancestors. He shows that it is possible that in cancer the reversion to the primitive cell type is conditioned by disordered bodily function. In other words, "the body has failed to carry out the contract under which the cell abandoned its right to unlimited multiplication."

In regard to lymph stasis, Handley points out that in order to produce cancer, the obstruction must be of long standing, it must have lasted twenty to thirty years. It may not be complete enough to cause obvious lymphatic œdema, but may manifest itself by papillary hypertrophy and increased cellularity of the subepithelial connective tissues. He calls to mind Warburg's work on the cell which indicated that the cancer cell is a cell which has somehow acquired an entirely new type of metabolism. He goes on to state that chronic lymphatic obstruction must profoundly influence the conditions under which the cell lives: the intercellular pressure will be raised, wandering cells brought by the blood will be trapped in the district and will be unable to leave it, the supply of oxygen will be restricted and the fluid bathing the walls will be relatively stagnant, lacking the supply of fresh hormones derived from other cells of the body which it should normally contain, and lacking specially the inhibitory hormone which is the sedative to the cell's primal passion for division. Later on in his article Handley explains that he uses the word hormone in a somewhat loose sense to signify those products which are necessary to the well-being of the cell. This growth restraining action of tissue fluid on the cell is, of course, purely hypothetical. The factor required in addition to chronic lymphatic obstruction, according to Handley, is chronic irritation; he states that Menetrier's belief that cancer is a morbid process resulting from "multiple and non-specific forms of irritation" is justified. Handley's view may thus be described in a few words as being the occurrence of malignant changes as a result of chronic irritation in a region which is already the site of chronic lymphatic obstruction.

[1] *The Canadian Medical Association Journal*, November, 1929.

In referring to Menetrier's work with X rays on guinea-pigs and the production in these animals of hyperplasia of the epithelium and metaplasmic alterations of the cells, Handley states that Menetrier described papillomatous appearances which he mistook as being due to the downgrowth of epithelial processes. Handley points out that in Menetrier's figures hypertrophied papillæ are to be seen in a stage of real, if incipient, papillomatosis. In the centre of each are cellular cords which Handley regards as being papillary lymphatics in a state of proliferative lymphangitis. The lymphangitis in his opinion is of primary importance and the epithelial hypertrophy is secondary, though he admits that a direct stimulating action of X rays on the epithelium doubtless occurs.

In any discussion on Handley's work it must be understood that he realizes that complete proof is not at present possible. He states that his views contain a large amount of speculation. At the same time he asks his readers to admit that lymph stasis lies at the root of cancer, certainly of carcinoma, probably of sarcoma. It will be generally admitted that the metabolism of the cancer cell is different from that of the normal cell and it will also be admitted that the normal cell which becomes cancerous, loses its normal growth restraint, for this loss of growth restraint is the inherent quality of the cancer cell. There is no proof that the growth restraint is the result of a substance carried to the cell by the pericellular lymph. The restraint or limitation of growth is generally spoken of as an inherent quality of the cell, for the reason that the mechanism of the restraint production or the nature of the substance producing the restraint is not known. The substance may be, as Handley suggests, carried to the cell by the intercellular substance or the growth restraint may be the result of a substance formed in the cell during the normal metabolism of the cells. It will be admitted, of course, that lymph stasis might act in a detrimental way to the metabolism of the cell and that the altered products of metabolism might interfere with the production of a growth restraining substance in the cells.

Obviously lymph stasis would not affect a single cell, but a group of cells. It will be remembered that Handley teaches that malignant disease spreads by lymphatic permeation. If this view be accepted, it will be clear in the light of his present communication why cancer is localized in its early stages and why dissemination occurs only after the primary growth has extended beyond a certain distance. If dissemination takes place by the lymphatics and if there is a preceding lymphatic obstruction, dissemination cannot occur until the original cancerous change has spread beyond the boundary of the obstructed area. Handley touches on this aspect when he discusses X ray epitheliomata. It is of interest also in that it illustrates the point that in the early stages cancer apparently spreads locally from cell to cell.

If Handley's views were accepted, it would be natural to expect to find cancer appearing in any condition in which lymph stasis was a prominent feature. Persons who suffer from chronic elephantiasis, would be expected to become the subjects of malignant disease as a result of chronic irritation. There is little evidence of much malignant disease among the native population of the islands of the Pacific who are notoriously affected by filariasis. Handley records the history of a patient with elephantiasis who was subjected to radiant heat baths and who manifested "virulent" multiple endotheliomata shortly afterwards. It is in the light of his hypothesis that he explains the occurrence of cancer in mice injected by Cherry with tubercle bacilli. He points out that tuberculosis is accompanied by lymph stasis and states that the specific factor is not the organism concerned, but the lymphatic obstruction to which the organism gives rise. He suggests that mild undetected tuberculous infection of the mammary glands in childhood may leave behind areas of lymph stasis which give rise in middle life to duct papilloma and carcinoma of the breast. It would be reasonable to expect that primary malignant disease of the lung would be much more prevalent in late adult life in view of the large number of healed tuberculous lesions of the lung occurring in adolescents. Handley goes so far as to state that if tuberculosis of the bowel does not result in an ulcer, it produces intestinal polyposis. Since papilloma is in his opinion the forerunner of carcinoma, carcinoma of the bowel in many instances would be traceable to early tuberculous infection—a new conception.

Another point worthy of consideration is that in operations for malignant disease the lymph drainage area extending well beyond the lesion is removed. This will result in more stasis and if Handley's views were correct, such patients, if they lived long enough, would be certain to suffer, not from metastases of the original growth, but from fresh malignant disease occasioned by the post-operative lymph stasis. Generally they do not live for the twenty or thirty years regarded by Handley as necessary for the production of cancer by lymph stasis.

Other aspects which could be discussed in the light of this work, are such questions as the effect of radium treatment and its relation to lymph stasis, the malignancy of a tumour and lymph stasis, X ray treatment for therapeutic purposes *et cetera*.

Discussion of this sort does not prove that Handley is right. It does not prove that he is wrong. It shows that though he has put forward a view which fits many of the facts and which coincides with his previous teaching, there is much to be determined. He suggests certain lines for research, such as the injection of intestinal lymphatics with tubercle bacilli. There is always the possibility that if lymph stasis is not the whole story, it may be an important part of it.

Abstracts from Current Medical Literature.

RADIOLOGY.

Pulmonary Tuberculosis of Infants and Children.

ROLLA G. KARSHNER (*American Journal of Roentgenology and Radium Therapy*, September, 1929) writing on pulmonary tuberculosis of infants and children, first describes the general Röntgen findings in pulmonary tuberculosis and states that the earliest lesions in adults occur as indistinct multiple shadows somewhere in an upper lobe. They are usually circular, variable in size, faint at first, but invariably surrounded by a zone of exudative inflammation. As healing occurs, the inflammatory zone disappears and the shadows become sharper. Caseation causes increased density. The next step is decrease in size and finally the larger lesions will manifest fibrosis or calcification. Three forms are thus met with: the proliferative form, the exudative form and the fibrous form. Calcification of tuberculous infiltrations is the best evidence of healing. Primary tuberculosis is classified under two headings, focal primary lesion and caseous pneumonia or infantile phthisis. A small primary focal lesion is seldom detected. The skiagram may reveal a pneumonic infiltration of moderate extent in any lobe; more frequently all that is seen are the healed remains of a small primary infection. Caseous pneumonia of tuberculous origin is not by any means always fatal. It may be primary or secondary from rupture of a caseous lymphatic gland into a bronchus. In tuberculosis of the intrathoracic lymphatic glands the diseased glands appear as dense shadows extending beyond the mediastinum into the pulmonary field. In infants this gland infection is relatively greater than in children, the mass often assuming the dimensions of a neoplasm. Glandular enlargement is usually greater on the right side than on the left. Tuberculous hilus glands may cause all the phenomena commonly associated with foreign body in the air passages. In older children the diagnosis of tuberculous hilus glands is more difficult and tuberculous glands cannot always be distinguished from moderate tumefaction of hyperplastic lymphatic glands, unless there is caseation or calcification. Thus in children the skiagram in hilus gland tuberculosis is of value, mainly to show whether there is actually pulmonary disease or whether the infection is confined to the bronchial glands, thus differentiating a grave condition from a benign one. In dealing with hilus tuberculosis, the author states that it is a much more common form of the disease in early than in adult life. In infants it consists of a cheesy tuberculosis of the lung root tissues, rapidly progressive, with a bad prognosis. Recurrent hilus pneumonia of tuberculous origin is characterized by a recurrence of symptoms of pulmonary infection at intervals of weeks or months and it affects both infants and children. The process is an exudative one, arising around the tuberculous lymphatic gland and if it resolves, the shadows almost completely disappear. Recurrences of such hilus pneumonia, though benign at first, may eventually be followed by a rapidly destructive involvement ending fatally. Basal tuberculosis without coincident involvement of the apices is a more frequent happening in early than in adult life. Miliary tuberculosis is a common terminal phenomenon in any type of tuberculosis. The first symptoms frequently follow closely upon measles or pertussis. The author's observations lead him to the belief that occasionally miliary tuberculosis heals, though proof of such phenomenon is difficult to adduce. As puberty is approached, the tendency is for adult forms of tuberculosis to develope. Lesions are more indurated and the apical regions more liable to be selected. Caseation vies with fibrosis; the result is the multiform picture of fibro-caseous tuberculosis with cavitation and the lobular pneumonia characteristic of the adult.

Classification of Chronic Joint Diseases.

R. KIENBÖCK (*Wiener Medizinische Wochenschrift*, December 21, 1929) following on his radiographic studies has attempted to classify chronic joint lesions. His first main group comprises trophic and nutritional disorders such as *arthritis deformans*, hæmophilic osteoarthritis, true gout, alkaptonuria, Heberden's osteoarthrosis and *arthritis ulcerosa sicca*. Included in this group are conditions of unknown origin such as Perthes's disease, Schlatter's disease and affections of the vertebral column, *os lunatum* and *os naviculare*. In addition he mentions the spinal arthropathies due to *tabes dorsalis*, syringomyelia and congenital diseases of the lower end of the spinal cord. The second main group includes lesions due to acute inflammatory disease as well as infections such as tuberculosis, syphilis, gonorrhœa and true rheumatic disease. The final section comprises tumour formations of innocent character, chondromata and solitary or multiple foreign bodies in joint cavities.

Gastric Ulcer.

FRANKLIN WHITE (*New England Journal of Medicine*, November 28, 1929) makes observations on the healing and on the immediate and late results of medical treatment in gastric ulcer. Fifty private patients with gastric ulcer have been under observation during six or eight years. The Röntgen rays have been of great value in diagnosis and by their means it has been possible to watch the healing of the ulcers and to judge the immediate results of medical treatment. In lesser curvature ulcers under treatment X ray examinations at intervals of two to three weeks show steady change and disappearance of the ulcer deformity and these changes coincide as a rule with the clinical improvement. They may be surprisingly rapid and large ulcers are cited in which extensive deformity may disappear in four to six weeks. In determining the risk of medical study and treatment two questions are to be decided; first, whether cancer is present and secondly whether cancer is present and second Accurate diagnosis in the beginning is the most vital single factor in eliminating the risk of medical treatment. No one factor in the diagnosis is entirely characteristic of cancer; neither the history, the gastric acidity, the occult blood nor the type of X ray deformity alone is sufficient, but all these factors taken together allow of very accurate diagnosis, especially if a probation period of several weeks is given and if during this time the effect of treatment is noted, both on the occult blood and especially on the X ray deformity. Simple ulcers usually manifest striking improvement. Gastric cancer is unlikely to change. In doubtful cases after this intense study exploration is undertaken. Some cancers and some ulcers have been excised, the latter perhaps needlessly, but the author states that as far as could be determined no cancer has been left which should have been removed. After discussing the usual objections raised to X ray evidence of healing of gastric ulcers, the author concludes that these are negligible. This series of cases offers good evidence that gastric ulcers actually heal in the intermission of symptoms and that healing may be very rapid, even in large ulcers of the lesser curvature. The age of the patient in the series has not especially influenced the healing. The size of the ulcer has little effect on the healing rate. Ulcers with four years' symptoms heal just as quickly as those with two months' symptoms.

Intracranial Disease.

JOHN D. CAMP (*Radiology*, December, 1929) in discussing Röntgenological interpretations of intracranial disease states that stereoscopic films, especially in the lateral projection, are essential. In addition antero-posterior and postero-anterior films are taken and frequently additional views showing the occiput, base, orbital areas and sinuses. The signs of intracranial disease in their order of importance are grouped as follows: Calcification within the lesion, localized changes in the bone, changes in the *sella turcica*, changes in the bone resulting from increased intracranial pressure, displacement of the pineal shadow, air in or about the lesion and increased vascularity of bone. The pineal gland, Pacchionian bodies and chorioid plexus may normally exhibit a certain amount of calcification. Pathological lesions that may exhibit areas of calcification, include tumours, hæmatomata,

tubercles, cysts, old abscesses, old meningeal lesions, encephalitis, sclerotic arteries and aneurysms. A partial differential diagnosis amongst these conditions is given. The bones of the skull overlying a tumour may manifest destruction, proliferation or both. The usual change is localized thinning of the inner table. Changes in the *sella turcica* may result from local or general increased pressure and are seen in the following conditions: Intrasellar tumours, both benign and malignant, metastatic tumours, gummata and tuberculomata; suprasellar lesions, not proper to the pituitary gland; disease of the sphenoid bone. Intrasellar tumours expand the fossa in a uniform circular manner at the expense of the floor and *dorsum sellæ*. Any cerebral neoplasm that produces an increase of intracranial pressure, will produce corresponding changes in the outline of the *sella turcica*. These changes are the result of transmitted pressure and consist of widening and flattening of the *sella turcica* with erosion of the clinoid processes from above. Disease of the sphenoid bone is usually accompanied by density of the sphenoidal sinuses and haziness of the floor of the *sella turcica*. When displacement of the pineal shadow is definite, this sign is very valuable for localization of a tumour and may save the patient a ventriculogram. Lesions which erode into the sphenoidal sinus, may allow air to enter the cranial cavity. Increased vascularity of bone must be interpreted with caution and, unless accompanied by evidence of erosion or other localizing signs, such changes are best ignored.

PHYSICAL THERAPY.

Röntgen Sickness.

A. SCHONIG (*Strahlentherapie, Band* XXXIII, 1929) has studied the effect of therapeutic Röntgen irradiation on the reticulo-endothelial system by means of the Congo-red method. There was a decrease in function of the reticulo-endothelial system immediately after irradiation with castration and carcinoma doses. It was found that the height of the Congo-red index and the intensity of Röntgen sickness ran parallel. If the reticulo-endothelial system absorbed the stain poorly, the sickness was more intense. If absorption of the stain was good, there was no Röntgen sickness. Apparently the absorptive capacity of the reticulo-endothelial system for Congo-red corresponds to its capacity for absorbing tissue toxins. Röntgen sickness is a symptom of intoxication of the body by products of disintegration of cells. Its intensity is dependent on the amount of products of disintegration produced by the action of the rays and the capacity of the reticulo-endothelial system for absorbing them. As Röntgen sickness is an evidence of serious changes in the defensive forces of the body, it should be avoided as much as possible. This can be accomplished by dividing the therapeutic doses into several fractional doses.

Second Toleration Doses in Röntgen Units.

P. M. HICKEY, E. A. POHLEA, G. A. LINDSAY, J. M. BARNES (*Radiology*, April, 1929) discuss the question of second toleration doses in Röntgen units and their relation to the quality of radiation. They suggest that for practical purposes in radiology the effective wave length should be employed as the quality factor. In any test the potential filtration and the transmitting material must be noted. Further studies of the relation of second toleration dose in Röntgen units and their relation to the quality of radiation have confirmed previous investigations. It seems safe to assume that small areas three centimetres by three centimetres may be exposed to 500 or 600 Röntgen units with all types of Röntgen rays used in practice. They examined a range from effective equal to 0·61 Ångström units to 0·138 Ångström units. They recommend that great caution be exercised in transferring these results to the exposure of large fields in daily practice because of the fact that there exists no simple method of measuring the exact amount of back scattering on the patient.

Tuberculosis of the Adnexa.

PAUL GIBERT (*Journal de Radiologie et d'Électrologie*, April, 1929) states that Röntgen therapy must be considered as of great importance in the treatment of tuberculous inflammation of the pelvic adnexa. Operation and procedure in these conditions have a mortality of 8%, whereas Röntgen therapy is entirely without danger. Furthermore, irradiation can be applied to even the most severe forms of the condition when operation is contraindicated. The irradiation consists in the application of fractional doses such as are generally used in tuberculous affection. Some Röntgen therapeutists consider that only temporary sterilization should be attempted, but the author is of the opinion that a permanent castration is of a greater value, because it is easier to carry out technically, because it will eliminate the congestion associated with menstruation which leads to exacerbation of the tuberculous process, and because pregnancy is not desirable in these cases. The dose consists in the administration of 500 Röntgen units (French) at each field once or twice a week, with one millimetre of aluminium and 0·5 millimetre of zinc until the total dose of 4,000 to 5,000 Röntgen units is reached. Two or three months later the series is repeated. Of seventeen patients treated, three died following other developments of tuberculosis, five manifested considerable improvement and nine were permanently cured, the period of observation being from one to four years. The author concludes that irradiation should be attempted in all tuberculosis of the adnexa and that only patients whose condition is refractory to this treatment, should be operated upon. In many cases conjoint use of operation and post-operative irradiation is of considerable value. The irradiation should be carried out with fractional doses until permanent menopause is obtained.

Radiation for Malignant Disease.

R. WERNER (*Strahlentherapie*, November, 1928) describes twenty far advanced malignant tumours in which radiation therapy has produced unexpected cure. The tumours cited are a carcinoma of the maxilla, healed for three and a half years; a carcinoma of the tongue with infiltration of the deep structures and extensive lymphatic gland metastasis, healed for eight and a half years; a carcinoma of the floor of the mouth with involvement of the submaxillary lymphatic glands, healed for six years; a recurrent melanosarcoma with extensive metastases, healed for five and a half years; a bronchiogenic carcinoma with metastasis to adjacent lymphatic glands, healed for ten years; a *struma magna*, with compression and severe respiratory disturbances, healed for four years; a periosteal sarcoma of the sternum, healed for eight and a half years; a spindle celled sarcoma of the upper arm, healed for eight years; a sarcoma of the upper arm in a hæmophiliac, healed for seventeen years; a recurrent mammary carcinoma with extensive skin metastases, healed for six years; a recurrent mammary carcinoma with metastases to the lumbar portion of the spine, healed for six years; a large mediastinal tumour with pressure symptoms, healed for eight years; a large mediastinal tumour with secondary stasis, healed for fourteen years; a recurrent carcinoma of the prostate with metastases to the cervical portion of the spine, healed for three years; a recurrent sarcoma of the inguinal lymphatic glands with extensive metastases, healed for nine and a half years; a lymphosarcoma of the inguinal lymphatic glands with metastases to the cervical lymphatic glands and again recurring ten and eleven years later, healed; a metastatic seminoma, healed for four years; a recurrent carcinoma of the ovary, healed for three years; a carcinoma of the sigmoid incompletely removed healed for eleven years and an inoperable carcinoma of the papilla of Vater still healed at the time of accidental death three years later. From this the author arrives at the conclusion that all malignant conditions should be treated systematically. It is impossible to foretell which of the tumours will respond to irradiation. Therefore even those entirely hopeless should receive a curative dose. Of course the general condition of the patient has to be taken into consideration and the dose administered accordingly. The administration of only a palliative dose in the conditions enumerated would have meant an incomplete procedure, most probably leading to death of the patient.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held at the Medical School, The University of Sydney, on August 29, 1929, Dr. F. Brown Craig, the President, in the chair.

The Origin and Spread of Cancer.

Professor D. A. Welsh read a paper entitled: "On the Origin and Spread of Cancer" (see page 282).

Dr. E. H. Molesworth said that the subject was too wide and too difficult for him to do justice to the occasion. Both the subject and the manner in which it had been presented, had been instructive and interesting. The problem of the genesis of cancer was intriguing and the more they looked into it, the more they realized how little they knew. The spread of the stimulus was real. It was mentioned in text books, but those who read the books, did not realize what was meant. It opened up a new field as to the origin of cancer. It was possible to imagine that the stimulus leading to cancer spread had originally been applied to normal cells and had caused them to become cancerous. Dr. Molesworth concluded by referring to Professor Welsh's collaboration at the Cancer Clinic. He always lifted the work on to a higher plane by the scientific value of his discussion. Dr. Molesworth was very grateful to Professor Welsh for his address.

Dr. F. A. Maguire expressed his appreciation of Professor Welsh's address. Professor Welsh had opened up new lines for contemplation and for observation. He asked Professor Welsh's opinion in regard to a condition which he had seen in two patients. One had been seen four years and the other twelve months after hysterectomy for malignant disease. In each instance a secondary cancerous growth had been present in the wall of the vagina and the structure had been adenocarcinomatous, similar in type to that of the tumour for which the uterus had been removed. Dr. Maguire asked why the cells should lie latent in the interstices of the vaginal wall, if they had done so, to come to light in one instance four years and in the other twelve months after operation. What was the stimulus which had caused them to take on growth or was there an infiltration or a change in the surrounding cells? In conclusion Dr. Maguire suggested that a post-graduate course in pathology would be valuable.

Dr. E. L. Morgan said that he had listened with great interest to Professor Welsh's address. He referred to the statement of Bland Sutton in regard to the secondary nature of tumours of the ovary. He agreed that ovarian tumours were as a rule primarily developed in that organ, but his experience had been different from that of Professor Welsh, for within two years he had seen two ovarian tumours which were secondary to tumours elsewhere. After examining one of them, a typical Krukenberg tumour, he had suggested that the growth was secondary to malignant disease of the stomach and further inquiry had shown that gastrectomy had been performed about twelve months previously.

Dr. Constance D'Arcy expressed her gratitude to Professor Welsh. She said that he had provided stimulus for the imagination and food for thought. She thought that if an obstetrician were to undertake the study of malignant disease, it might be given to him to discover something about the origin of cancer. In the parturient female they had exemplified the results of the stimulation of cells to growth in an orderly fashion, resulting in the formation of a fœtus. Wild growth was exemplified in the placenta. The same stimulus caused an increase of cells in other parts of the body, for example, the thyreoid, the mammary and the suprarenal glands. There was to be considered the secret of the factors which stopped the wild growth. The placenta penetrated the uterine wall, but some factor kept it from indefinite spread. Dr. D'Arcy in conclusion referred to the lectures which had been given by Professor Welsh under the ægis of the Cancer Research Committee, and expressed the hope that further lectures would be arranged.

Dr. F. Brown Craig thanked Professor Welsh for his brilliant and stimulating address and for the fascinating way in which he had presented the subject. They were grateful to Professor Welsh for having allowed them to hold the meeting at the medical school while they were temporarily without a home.

Professor Welsh in reply said that he appreciated the way in which they had received his effort. It had been an attempt to give expression to the experiences of many years and it had been impossible in the time at disposal to do the subject full justice. The question of long latency referred to by Dr. Maguire was one of the inexplicable problems of cancer. The growths to which Dr. Maguire had referred, had been due to cancer cells which had lain dormant for a long time. It was not so much a question of what stimulus had caused them to grow, but rather of what influence had been exerted on them for four years to keep them quiet. He referred to examples of the same sort of latency which were sometimes seen in cancer of the breast when a secondary growth would be found after considerable time in the vertebral column or when a spontaneous fracture due to metastases occurred in the femur. He had been interested to hear Dr. Morgan's remarks. All he could say was that ovarian tumours of such a nature had not come within his experience. Dr. D'Arcy had been quite right in what she had said. The idea of newgrowth found its nearest approach in the behaviour of the trophoblast in relation to the decidua. It was a tumour of local, limited and regulated malignancy.

A meeting of the Obstetrical Committee of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on July 23, 1929, Dr. Alex Marks, the President, in the chair.

Adoption of Report.

The quarterly statistical report was read and adopted and certain selected case reports were read and discussed.

Acute Yellow Atrophy of the Liver.

Dr. F. A. Hope Michôd reported a death from acute yellow atrophy of the liver. A *primipara*, aged twenty-nine, had estimated the date of her confinement as May 10, 1929. The first antenatal examination had been made on December 4, 1928. She had been seen every fortnight between March 3, 1929, and the date of the birth. The urine had been examined on every occasion and no albumin had been detected at any examination. The patient had apparently been a perfectly healthy woman. There had been no history of any previous illness and the possibility of a previous septic focus or of lead poisoning had particularly been investigated. She had camped out with her husband in the bush between December 4, 1928, and March 11, 1929. Labour had commenced at 9 a.m. on May 13, 1929. The first and second stages had lasted forty hours, the pains had been weak and a deficiency of *liquor amnii* had been noted. She had been delivered of a full term healthy child, weighing 3·8 kilograms (eight and a half pounds); labour had been terminated with the assistance of "low" forceps. The patient had been kept under the influence of hyoscine from 3.30 p.m. on May 13 until 6 p.m. on May 14. The first injection had consisted of morphine, 0·016 gramme (one-quarter of a grain), hyoscine, 0·65 milligramme (one one-hundredth of a grain), atropine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), and this had been followed by thirteen injections of 0·16 milligramme (one four-hundredth of a grain) of hyoscine. At the commencement of the administration the fœtal heart rate had been 148 and the maternal pulse rate 88 in the minute and during the whole period the fœtal heart rate had varied between 148 and 160 and the maternal pulse rate between 80 and 92. At 11 p.m. on May 14 the patient had been given a few drops of chloroform during a pain and this had been continued until the child was born at 12.50 a.m. on May 15. The

patient's condition had never been that of full surgical anæsthesia, except possibly for about ten minutes at 12.30 a.m. while instruments were being applied.

On May 15 at 10 a.m. the patient had been very well and had said that she felt fit to get up. The pulse rate had been 80 in the minute and the temperature 37·4° C. (99·4° F.). She had passed urine naturally, nothing abnormal had been noted in her appearance and she had had a light luncheon at 1 p.m. The baby had been put to the breast at 2 p.m. The patient had immediately felt a pain in the epigastrium and the right hypochondriac region; her stomach had been dilated and she had brought up a lot of wind. The pulse rate had been 80 in the minute and the temperature 37·1° C. (98·8° F.). The ordinary remedies for flatulence had been given and on account of the pain 0·016 gramme (one-quarter of a grain) of morphine. There had been no vomiting.

On May 16 it had been reported that the patient had passed a fair night, had slept most of the time and complained of slight pain in the region of the liver. The pulse rate had been 84 with the temperature 36·7° C. (98° F.). The patient had been deeply jaundiced. It had been impossible to define the lower border of the liver. Urine had been scanty, dark red and no leucin or tyrosin crystals had been detected and no acetone. The patient had begun to eject small quantities of brown stained fluid. A few scattered petechiæ had been found scattered about the trunk and extremities. The patient had become drowsy.

On May 17 the patient had been worse. The pulse rate had been 96 and the temperature normal. Jaundice had been more pronounced, the urine not so deeply coloured with blood, but very scanty. The patient had become more and more drowsy, had commenced to vomit quantities of black vomit, drowsiness had increased until she passed into a state of coma and died at 2 a.m. on May 18, seventy-four hours after the birth of the child.

Dr. Michôd said that the following points were of note. The patient was a particularly healthy woman, the confinement was somewhat prolonged, but otherwise normal. A perfectly good recovery had occurred immediately after the birth of the child. Thirteen hours after the delivery there had occurred the sudden onset of very severe pain in the hepatic region with some gastric distension. Jaundice had been noticed at daybreak the following day and it had become deeper and deeper. No vomiting had occurred until a few hours before death, but an eructation of a brown coloured fluid had commenced on the afternoon of May 16, about thirty-eight hours after the birth; this brown coloured fluid had soon become distinctly "coffee-ground" in character. The patient had become drowsy towards the end of the second day, becoming more and more comatose and passing into deep coma just before death. The temperature had been raised at the end of the confinement, but had come down in the first twenty-four hours and remained subnormal. The pulse rate had increased to 124 just before death.

At *post mortem* examination the liver had appeared shrunken and histological examination revealed an extensive necrosis of liver cells with no deposit of fat.

As a result of the clinical and *post mortem* report Dr. Michôd considered that the condition was one of acute hepatic necrosis, due to toxæmia of pregnancy, the differential diagnosis being between acute necrosis and delayed chloroform poisoning. As both conditions were so rare, he had looked up the literature on the subject, with the following result. Osler and McCrae in the 1926 edition of their system described the disease as "an acute, widespread, autolytic necrosis of the liver cells, characterized clinically by jaundice, reduction in the size of the liver and a toxic disturbance of cerebration, proceeding to a fatal issue." The incidence was one in 280,000 confinements.

De Lee stated that the condition might occur at any stage of gestation or during the puerperium. It was frequently the termination of *hyperemesis gravidarum*, eclampsia, chloroform poisoning and it was sometimes epidemic. At autopsy a wasted liver of a yellow colour with albuminoid degeneration of liver cells was found. The symptoms were anorexia, constipation, headache, a slight mental disturbance, extreme weakness, icterus, pain in the epigastrium, vomiting which steadily increased, became bilious, then bloody. The patient became delirious, then passed into a state of coma and died. There might be petechiæ present and slight cyanosis. The pulse rate varied from 120 to 140 and the temperature was in the neighbourhood of 38·9° C. (102° F.). There was a diminution in the quantity of urine. Leucin and tyrosin crystals were present.

Vaux emphasized the "black vomit" and stated that in severe cases there might be no period of insomnia, but the patient passed directly into a comatose stage and usually died in a few days, in very acute cases in forty-eight hours.

William emphasized that in mild cases the centre of each lobe might have undergone necrosis while the cells of the periphery of each lobe were normal. In more severe cases almost the entire parenchyma of each lobe was destroyed and was converted into a granular mass of necrotic *débris*, while at the periphery a few normal liver cells might be observed. It might occur at any time during pregnancy or during the first few days of the puerperium. The symptoms were a sudden onset (a woman in apparently perfect health might be seized with pain in the abdomen, intense headache, vomiting and purging, in a short time she became torpid and soon passed into a condition of coma), jaundice, coffee-ground vomit, urine diminished in amount and frequently containing a large quantity of blood.

As a result of a short *questionnaire* sent out to all the members of the Branch the following replies had been obtained.

Sir David Hardie had forwarded a copy of a paper he had read before the Medical Society of Queensland on March 11, 1890, in which he gave an account of six cases which occurred in his practice during the previous eight months.

The first case had occurred in a house where Sir David Hardie was attending a man who was suffering from ordinary catarrhal jaundice. The woman had been twenty-eight years of age and had had two children. When seven months pregnant she had become jaundiced and the condition had been looked upon as one of ordinary catarrhal jaundice similar to that of the man. Seven days after the onset of the jaundice the woman had become drowsy, for eight days she had been semicomatose and had been delivered of a premature child which was jaundiced, but lived for seven days. After nine days profound coma had occurred. The pulse rate had been 115 and the temperature 38·9° C. (102° F.). Diminished liver dulness had been present. Urine had not been scanty. The patient had died on the tenth day after the commencement of the jaundice, three days after the premature delivery. At *post mortem* examination the liver had been found reduced to half its normal size. The spleen had been pulpy, the kidneys healthy. No examination had been made for leucin and tyrosin.

Sir David Hardie's second patient had been twenty-five years of age, had had two children and had been six months pregnant. A consultation had been held because the urine was dark in colour and the patient, slightly jaundiced, had complained of giddiness, epigastric pain and frequent vomiting. Examination had revealed diminished liver dulness, urine with a specific gravity of 1010 and containing bile and a trace of albumin. No leucin or tyrosin crystals had been present. On the following day violent delirium, uterine hæmorrhage and a miscarriage had occurred. The patient had been comatose. The temperature had been 38·6° C. (101·5° F.) and the pulse rate 120 in the minute. Leucin had been present. The patient had passed into profound coma and had died three days after the onset of serious symptoms.

The third patient had been six months pregnant. She had died twenty-four hours after the onset of serious symptoms.

The fourth patient, aged twenty-one years, had had two children and was a strong, muscular woman. She had been four months pregnant, semicomatose and jaundiced. The pulse and temperature had been normal. Liver dulness had extended five centimetres (two inches) above the costal margin. The urine had been dark for the previous ten days. The patient had been attending to her household

duties on the previous day. On the evening of the same day deep coma had occurred. The respirations had numbered 45 and the pulse 96 in the minute. The temperature had been 37·8° C. (100° F.). Rigidity of the muscles of the upper and lower extremities had been present and the patient had died on the following morning. *Post mortem* the liver had been rhubarb yellow in colour, soft and friable, it had weighed 905 grammes. The spleen had been soft and the kidneys normal.

The fifth patient had been twenty-seven years of age, had had three children and had been pregnant seven months. Sir David Hardie had been sent for on account of severe epigastric pain, slight jaundice and constipation. Liver dulness had been 2·5 centimetres (one inch) above the costal margin. For three days the patient had attended to her household duties. The specific gravity of the urine had been 1026. No leucin or tyrosin crystals had been found. On the fifth day coffee-ground vomiting had occurred. On the sixth day the patient had been drowsy. The pulse rate had been 84 and the temperature normal. Liver dulness had extended five centimetres (two inches) above the costal margin, constant vomiting had been present. The urine had had a specific gravity of 1018; it had contained a trace of albumin, bile pigment and leucin and tyrosin crystals. On the evening of the sixth day the patient had been restless and delirious. The child had been born at 11 p.m. The *liquor amnii* had contained leucin and tyrosin crystals. On the seventh day the patient had been fully comatose in the morning. The pulse rate had been 124, the respiratory rate 52 and the temperature 38·6° C. (101·5° F.). Rigidity of muscles of the extremities had occurred and the patient had died the following morning, on the eighth day after the onset of symptoms. *Post mortem* the liver had weighed 849 grammes; it had been in an advanced stage of fatty degeneration. Microscopically the liver cells had been almost entirely destroyed; fatty globules had been present.

The sixth patient had been three months pregnant. She had complained of headache and giddiness and epigastric pain and jaundice for ten days with constipation. Three days later violent delirium had occurred. The pulse rate had been 130 and the temperature 37·2° C. (99° F.). Abortion had occurred. Liver dulness had been reduced two fingers' breadths, coffee-ground vomiting had occurred the day before. The specific gravity of the urine had been 1018. It had contained no albumin. Leucin and tyrosin crystals had been present. At 8 p.m. on the third day the patient had been deeply comatose. Her pulse rate had been 120, her temperature 38·3° C. (101° F.) and her respirations had numbered 40. No rigidity of muscles had been present. She had died at 11 p.m. on the third day. *Post mortem* the liver had been yellow, soft and friable; it had weighed 849 grammes. Fatty degeneration had been present in liver and spleen and to a less degree in the kidneys.

Sir David Hardie had said that he had not seen a case before the first one reported; all six cases occurred in Brisbane during eighteen months and he had not seen a case since.

Dr. G. A. Sampson had reported a case which had occurred in the Lady Bowen Hospital on May 26, 1914. Eclampsia had been followed a week later by acute necrosis of the liver. The patient, aged twenty-five years, had had two children and had been eight months pregnant. She had suffered in turn from eclampsia, fits and coma. Dietetic treatment with eliminatives had effected improvement. Five days later epigastric pains, vomiting and hæmatemesis had occurred. Six days later jaundice had been noted. The temperature had been 38·9° C. (102° F.) Delirium had occurred and the patient had been confined of a dead child. Suppression of urine and hæmaturia had followed. Seven days later muscle twitching and petechiæ had been noted and constant pain had been present in the upper part of the abdomen. The patient had died at 2.30 p.m. No chloroform had been used.

Dr. E. S. Meyers had reported a case associated with pregnancy. The patient had had one child. No pain had been present, but jaundice had been noted. Albumin had been present in large amount; none had been found on previous examination. Vomitus had been dark and foul smelling. The patient had become comatose and had died three days after delivery. Chloroform had been administered on several occasions during labour and instrumental delivery had been carried out.

Notes had been submitted by the Assistant Matron of the Lady Bowen Hospital of a case which occurred at Esk. The patient had been a *primipara*, aged thirty-two and had been seven months pregnant. She had been admitted to hospital complaining of severe abdominal pain. Premature labour had occurred on the following day at 5.30 p.m. At 8.30 p.m. on the same day she had commenced vomiting copious coffee-ground material. Next day, in the morning, distressed breathing with slight jaundice had occurred and in the afternoon she had become comatose. At 10 p.m. deep coma had occurred. The urine had contained "one quarter" albumin. She had died at 10 a.m. on the following day, deeply jaundiced, forty hours after confinement. No albumin had been detected during pregnancy, although frequent urinary examinations had been made.

Dr. Ellis Murphy had reported four cases of acute hepatic necrosis, but three of them were not connected with pregnancy and the fourth condition was probably one of hepatitis in which recovery occurred.

Dr. Michôd said that he had searched for information about delayed chloroform poisoning and had found that the "Medical Annual" of 1909 contained a *résumé* of a paper read by Guthrie before the Society of Anæsthetists on delayed chloroform poisoning. Guthrie had stated that symptoms did not appear until twelve to thirty-six hours had elapsed. The patient became wildly excited, uttering piercing cries, consciousness might be lost and never regained. Delirium might alternate with drowsiness. Eyes were sunken, pupils dilated, face haggard and abdomen retracted; coma might supervene. The patient did not complain of pain. Jaundice was uncommon, but when it was present, the symptoms were those of acute yellow atrophy. Vomiting was the earliest and most constant symptom; it became "coffee-ground" in character and urine was always scanty. Acetone and diacetic acid were always present. The condition might prove fatal in twelve hours, usually in three to four days. *Post mortem* the liver was strikingly pale, might be canary or fawn coloured, streaked with minute purple streaks and lines. It was intensely fatty.

To sum up, the symptoms were those of acute fatty intoxication and *post mortem* extensive fatty necrosis of liver cells was found.

In the discussion which followed Hunter had pointed out that though the symptoms of acute fatty intoxication were somewhat similar to those of acute yellow atrophy of the liver, the pathological changes were distinct, for in acute yellow atrophy there was not a deposit of fat, but complete necrobiosis.

In regard to delayed chloroform poisoning, Dr. Michôd referred to cases reported in *The Lancet* of February 29, 1908.

A child, aged two and a half years, had suffered from tuberculous disease of the testicle. He had been operated on on December 3. The child had recovered promptly from the anæsthetic, but had started vomiting on December 6. Coffee-ground vomit and restlessness had been noted, but no jaundice or cyanosis. The temperature had been 36·7° C. (98° F.). The pulse had been small and thin; it had been impossible to count it. The patient had died on December 7. *Post mortem* the liver had been bright yellow, greasy and obviously in a condition of extreme fatty change. Microscopical examination had revealed liver cells with signs of very advanced fatty degeneration, in greatest amount at the periphery of the lobules.

A girl, aged eight years, had been operated on on December 16, 1907, for double osteotomy of both femora. The operation had lasted twenty-five minutes. In the evening she had been restless and screaming. On December 17 constant vomiting had occurred and diacetic acid had been present in the urine. Black material had been vomited. No jaundice had occurred and the patient had died on the morning of December 18. *Post mortem* the liver had not been enlarged, it had shown signs of extensive fatty change. Microscopically fatty cells had been most prominent in the peripheral cells of the lobules.

Dr. Hugh G. Wilson had reported a similar case in a child aged six and a half years. *Post mortem* the liver had been enlarged. Microscopically the liver cells had shown extensive and advanced fatty infiltration, but no degeneration of cell protoplasm.

As these cases were of such extreme rarity, but at the same time of considerable interest, it was thought that a discussion on the subject might be of interest to the members. Apparently in both conditions symptoms came on within a few hours or at most two or three days from the labour or administration of chloroform. The chief features might be set out in tabular fashion as follows:

Acute Hepatic Necrosis.	*Chloroform Poisoning.*
Jaundice pronounced.	Jaundice exceptional.
Epigastric pain constant.	No complaint of epigastric pain.
Flatulence, then vomiting, not distressing, but eventually becoming more severe and finally "coffee-ground" in type.	Vomiting most pronounced and a constant symptom, rapidly becoming "coffee-ground" in type.
Leucin and tyrosin crystals present in the urine.	No leucin or tyrosin crystals in the urine, but acetone and diacetic acid.
Drowsiness, passing into coma.	Delirium, restlessness, coma.
Post mortem, small liver, deep yellow. Necrosis of the cells, most pronounced in the central cells around the portal vein.	Liver not diminished in size, often enlarged, light yellow, greasy and fatty. No necrosis of liver cells, but extensive deposit of fat in the peripheral cells of the lobules.

A condition that was sometimes mistaken for acute necrosis of the liver was jaundice developing late in the puerperium. This was much more likely to be due to a cholangitis, due to sepsis, and if pain were present, it was probably due to a septic embolism in the liver.

Dr. Duhig had prepared sections of the liver in this case and pictures of these were thrown on the screen. Dr. Duhig explained the points of interest.

Imperforate Anus.

A case of imperforate anus was reported. An unsuccessful attempt had been made to remedy the condition, the child dying at the end of a week.

Placenta Prævia.

The history of a *primipara*, aged thirty-five, with *placenta prævia* (central) was read. This patient had had severe hæmorrhages at the fifth month and at six and a half months, followed by frequent small losses. Cæsarean section had been performed, the child unfortunately living for only five hours.

Acute Pulmonary Œdema.

A case of acute pulmonary œdema with recovery of the patient was reported by Dr. J. A. Cameron. In the discussion a case was quoted of recovery from this condition in the first confinement with recurrence and death in the second.

DR. H. S. WATERS had seen œdema just before labour which recommenced *intra partum*; the patient had died.

DR. M. H. ELLIOT SMITH had seen one example of the condition in a patient who died twenty minutes after onset, treatment being by atropine and venesection.

Induction of Labour.

The question of the efficiency of injections of ovarian residue for the induction of labour was raised. The consensus of opinion among those members who had tried it, was that it was successful in about 25% of patients at full time.

MEDICO-POLITICAL.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on February 5, 1930, DR. J. NEWMAN MORRIS, the President, in the chair.

DR. C. H. MOLLISON, the Honorary Treasurer, presented the financial statements of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria. The statements were received and adopted (see pages 300, 301).

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Chapman, Alick Ian, M.B., Ch.B., 1928 (Univ. Melbourne), Children's Hospital, Carlton.

Rutherford, Gideon McCrae, M.B., Ch.B., 1928 (Univ. Melbourne), 22, Bellair Street, Kensington.

Sheppard, Frederick Augustus Berrill, M.B., Ch.B., 1929 (Univ. Melbourne), 7, Kensington Road, South Yarra.

Turner, Edwin Watchorn, M.B., Ch.B., 1928 (Univ Melbourne), Alfred Hospital, Prahran.

LISTS OF MEMBERS.

THE lists of members of the several Branches of the British Medical Association in Australia are now in the press and will be available in a short time. Copies can be purchased from the office of THE MEDICAL JOURNAL OF AUSTRALIA at one shilling each.

Medical Societies.

PUBLIC MEDICAL OFFICERS' ASSOCIATION.

PUBLIC medical officers to the number of thirty-two met in the Assembly Hall of the Education Department, Sydney, on the afternoon of September 2, 1929, at the invitation of the Public Medical Officers' Association of New South Wales.

DR. E. S. STOKES took the chair and extended a hearty welcome to the visitors from the other States. He said that the main objects which the Public Medical Officers' Association of New South Wales had in view in arranging the meeting, were two. The first was to enable public medical officers from various States to meet and to know each other. In furtherance of this object the association had also arranged a motor car excursion for September 9, when he hoped as many inter-State visitors as could be present would be the guests of the association. It was generally agreed that cooperation amongst the public medical services was necessary and this would be made more easy if medical officers came to know and understand each other.

The second object of the meeting was to discuss some matters of common interest and he called upon the Honorary Secretary of the Public Medical Officers' Association of New South Wales to speak of the action taken in recent years to advance the interests of public medical officers in that State.

DR. H. HASTINGS WILLIS gave a brief outline of the movement which had resulted in the formation of a public medical officers' association in New South Wales in 1926, and a brief summary of that association's main activities since, especially stressing its struggle for better remuneration for its members and for facilities for post-graduate study.

VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Statement of Receipts and Payments for Period from January 9, 1929, to January 9, 1930.

RECEIPTS.	£ s. d.	£ s. d.
January 9, 1929.		
To Balance, National Bank of Australasia, Limited	504 18 4	
„ Cash in Hand	12 0 0	
		516 18 4
January 9, 1930.		
„ Subscriptions—		
Metropolitan Members	3,443 17 0	
Country Members	1,713 15 8	
Junior Members	374 11 0	
Capitation Grant, London	0 12 0	
	5,532 15 8	
Less Refund of Subscription	1 0 0	
		5,531 15 8
„ Clerical Work for Members	52 16 9	
„ Clerical, Permanent Post-Graduate Committee	25 0 0	
		77 16 9
„ Refund Expenses *re* Hall		24 10 0
„ Sale, Hospital Report Forms		0 4 0
		£6,151 4 9

PAYMENTS.	£ s. d.	£ s. d.
January 9, 1930.		
By Medical Society of Victoria		2,734 5 6
„ *The British Medical Journal*		1,700 10 0
„ Federal Committee		135 14 0
„ Salaries—		
Secretary	27 0 0	
Assistant Secretary	258 18 4	
Library Assistant	169 10 10	
Junior Clerk	79 2 6	
		534 11 8
„ Rebates to Subdivisions		96 10 0
„ Postages, Duty Stamps *et cetera*		92 0 11
„ Printing		114 6 2
„ Stationery		73 12 6
„ Commission on New Members		7 15 0
„ Advertising		10 14 0
„ Audit Fee, 1928		5 5 0
„ Telephone		10 14 6
„ Electric Light and Power		17 11 3
„ Legal Expenses		4 4 0
„ Repairs *et cetera*		5 14 9
„ Bank Charges *et cetera*		19 19 3
„ Insurance		1 1 7
„ Clerical Assistance	0 11 0	
„ Travelling Expenses	0 4 0	
„ Expenses—		
Hamilton Meeting	3 13 0	
Sir Ewen Maclean's Visit	5 17 5	
Yallourn Meeting	3 0 0	
Bendigo Meeting	2 6 0	
„ Lanternist, Branch Meetings	17 5 6	
„ Hire of Chairs	2 3 0	
„ Afternoon Teas	1 2 0	
„ Acts, Regulations *et cetera*	2 3 6	
„ Buglers, Memorial Service	2 0 0	
„ Binding Minutes	1 15 0	
„ Framing Photographs	8 15 6	
„ Photographs	1 14 6	
„ Wreaths	5 5 0	
„ Sundries	4 8 5	
		62 3 10
„ Balance, National Bank of Australasia, Limited	512 10 10	
„ Cash in Hand	12 0 0	
		524 10 10
		£6,151 4 9

Compared with the Books and Accounts of the Victorian Branch of the British Medical Association and found to be in accordance therewith.

C. STANTON CROUCH,
Secretary.

CRAWFORD H. MOLLISON,
Honorary Treasurer.
Melbourne, January 16, 1930.

J. V. M. WOOD & COMPANY,
Chartered Accountants (Australia).

The President said that no definite agenda paper had been prepared, but he knew that some matters would be submitted for discussion. He was willing to receive motions and would give priority to those submitted by the visitors.

The Secretary stated that letters of invitation to the meeting had been sent to the heads of all medical services in all the States and in New Zealand and suggestions for the agenda paper had been asked for. He briefly summarized the replies received from the Chief Quarantine Officer in Western Australia, the Inspectors-General of Mental Hospitals in Victoria and Western Australia, the Medical Officer of Health of Adelaide, the Senior Medical Officers of the Repatriation Department in Western Australia and in South Australia, the Principal Medical Officers of the Education Departments of Tasmania, Victoria and Queensland. Other replies were formal acknowledgements.

Post-Graduate Study.

DR. KENNETH SMITH moved:

> That it is to the best interest of public medical officers and of the community that post-graduate study should be compulsory and that the cost should be defrayed by the employing authority.

He said the purpose of the motion was to permit of a general discussion of the subject of post-graduate study. It was to the interests of the community in general that public medical officers should be as efficient and as up to date in the knowledge of their particular specialty as possible and to insure this post-graduate study was

MEDICAL SOCIETY OF VICTORIA.

Statement of Receipts and Payments for Period from January 9, 1929, to January 9, 1930.

RECEIPTS.	£ s. d.
January 9, 1929.	
To Balance, National Bank of Australasia, Limited	25 5 1
January 9, 1930.	
„ Subscriptions	2,735 0 6
„ Transfer from Medical Agency on Account of Building Fund	210 17 6
„ Sundries	2 6 5
„ Balance, National Bank of Australasia, Limited	92 16 9
	£3,066 6 3

PAYMENTS.	£ s. d.	£ s. d.
January 9, 1930.		
By THE MEDICAL JOURNAL OF AUSTRALIA		1,633 11 0
„ Interest on Building Fund Debentures		426 8 4
„ Redemption of Building Fund Debentures		100 0 0
„ Salaries—		
Secretary	412 0 0	
Library Clerk	71 0 0	
Caretaker	100 0 0	
Library Assistant	6 10 0	
Bonuses to Staff	22 10 0	
		612 0 0
„ Library, Journals and Binding		29 9 8
„ Furnishings		31 13 0
„ Postages		92 0 10
„ Rates, Taxes and Insurance		42 19 9
„ Light and Power		17 11 3
„ Audit Fee, 1928		5 5 0
„ Bank Charges		0 10 0
„ Telephone		10 14 6
„ Repairs—		
Electrical	5 14 3	
Plumbing	3 2 6	
Furnishings	24 16 6	
Building	25 13 8	
		59 6 11
„ Sundries		4 16 0
		£3,066 6 3

Compared with the Books and Accounts of the Medical Society of Victoria and found to be in accordance therewith.

C. STANTON CROUCH,
Secretary.

CRAWFORD H. MOLLISON,
Honorary Treasurer.

Melbourne, January 16, 1930.

J. V. M. WOOD & COMPANY,
Chartered Accountants (Australia).

essential. To the individual such study was of great advantage and the Federal Arbitrator had embodied this principle in his award for the Federal health officers by fixing an efficiency bar at the salary of £852 *per annum*, advancement beyond that rate being contingent on a medical officer having undertaken a course of study and passed such examinations as were required by the head of his department, for example, the diploma of public health and the diploma of tropical medicine. All facilities for obtaining these diplomas were given by the employing authority. Services such as the Royal Army Medical Corps and other British services had made post-graduate study obligatory and gave all facilities.

The Public Medical Officers' Association of New South Wales felt very strongly on this subject and hoped to see the introduction of systematic courses of post-graduate instruction in all branches of medical work by the Australian universities, so that facilities equal to those in Great Britain would be available to Australians and medical men in the far east.

He thought that there should be no promotion to senior or administrative positions unless the medical officer could produce evidence of post-graduate study in his particular specialty. The word costs in the motion stood for both time and money.

DR. E. H. RUTLEDGE seconded the motion.

DR. JANE GREIG expressed her pleasure at being present and congratulated the association in New South Wales on its activities. In Victoria their association had not been able to do as much for them as they would have liked, but she looked forward to better things in the future. She thought that post-graduate study was desirable for medical officers, but her experience as an administrator had been concerned more with the difficulties than with the advantages of it. She had been able to obtain half-time leave without pay for one member of her service and this had enabled him to obtain a diploma in public health, which could be obtained in Melbourne only after a rather long course of study of subjects many of which had little bearing on the work of a school medical officer. Unfortunately, this officer, as soon as he had obtained the diploma of public health, had resigned to take a more remunerative position elsewhere. Hence she was inclined to think that if concessions or privileges were allowed, some bond should be entered into and some assurance given by the medical officer that he would remain in the service for a specified time before he was given any study leave. Moreover, she doubted whether it was really worth while to give study leave to very junior members of a medical service, though for seniors such a course of study was invaluable and if in another country had many additional advantages. She thought that facilities for this would result in considerable advantage to the public medical services.

The motion was carried.

DR. EVAN JONES moved:

> That this conference is of opinion that public medical officers' associations should be formed in all the Australian States and to further this object appoints an organizing committee, consisting of public medical officers.

He thought that all would agree that it was to the public advantage that the best type of graduates should be attracted to the public service; to insure this it was

necessary that the conditions of service should be as attractive as possible. The most effective and convenient way of doing this was to form public medical officers' associations in all the States. Apart from this, for special reasons of its own, the body in New South Wales desired to see a great improvement in the rate of remuneration of medical officers in the other States. From his experience last year as a negotiator with the New South Wales Public Service Board he knew how necessary it was for salaries to be increased in all the States. Had the salaries of medical officers in some of the other States not been so low, they would have gained greater increases in New South Wales. There was a tendency to compare salaries in the various States and in this way a low salaried service was used as a reason for refusing more generous salaries in other services. He hoped to see associations formed in all the larger Australian States and such a committee as he suggested would be useful in effecting this.

Dr. Hastings Willis said that he was anxious to second the motion, because he thought the formation of associations in all the States would be of benefit to all concerned and indirectly to the general public. He drew attention to the second clause of the motion. It had been suggested to the New South Wales committee in 1928 by an eminent hygienist since deceased, that a Federal organization with Branches in the States would be preferable to a number of State associations. There was much to be said for such a plan, but there were great objections to it, some of which he stated. In fact, the Public Medical Officers' Association of New South Wales was strongly opposed to such a scheme and preferred to see in each State a separate organization with its own constitution, suited to its own needs and pursuing its objects in its own way. To insure cooperation amongst such associations some central committee would be necessary and he thought that the members assembled would be a suitable body to appoint it. Moreover, this committee could arrange for future conferences unless it was thought better to leave that duty to the association in the State where the medical congress was held. He hoped that the meeting would be the first of many and he would like to see some committee definitely charged with the responsibility of calling members together.

Dr. C. A. Courtney expressed his pleasure at being present and acknowledged the warmth of the welcome given to inter-State visitors. In his home State, Victoria, the activities of the Public Medical Officers' Association of New South Wales and its good results were well known and even envied; this made the organization in Victoria hopeful. Public service medical officers certainly had special interests that needed watching and the formation of organizations such as those contemplated, was highly desirable in the interests of the public and of the medical officers. Though resident in Victoria, his work frequently took him to all States, as his department was a Federal one; from the knowledge he had gained of the varying conditions he felt that there was useful work for public medical officers' associations in all States. He heartily supported the proposal.

Dr. Oliver Latham said that the motion went a little too far. He did not think the time was opportune for the organization of any Federal medical officers' association. There was much to be done to secure the formation of associations in the various Australian States; efforts should be directed to that end. When associations had been formed and were flourishing in all the States, the question of a coordinating committee such as that suggested could be considered by delegates duly appointed for that purpose by the various associations. He thought it a mistake to attempt too much at this stage.

Some informal discussion ensued, as a result of which it was agreed to delete the latter portion of the motion dealing with the appointment of a committee. The first portion of the motion was then carried, it being understood that the Public Medical Officers' Association of New South Wales would endeavour to organize a similar association in Queensland and in Western Australia and that the question of the next conference of public medical officers in Australia be allowed to stand over for a time.

Post-Graduate Work.

COURSE IN APPLIED PATHOLOGY IN MELBOURNE.

The Melbourne Permanent Committee for Post-Graduate Work announces that a special course of ten lecture-demonstrations on recent advances in applied pathology will be given at the University of Melbourne by Professor P. MacCallum in April, May and June, 1930. The lecture-demonstrations will be delivered each Tuesday at half past eight o'clock in the evening. The fee for the course is three guineas. Those who intend to participate are requested to send their names to the joint honorary secretaries, Dr. Harold Dew and Dr. W. S. Johnston, 12, Collins Street, Melbourne, as soon as possible and to remit the amount of the fee. Further particulars will be forwarded on application. The following is a summary of the course. The date of the first lecture has been altered.

Tuesday, April 1, 1930.

The blood and hæmopoietic tissues; the nature and relationships of the reacting tissues; the balance of blood cell formation and destruction; differential cellular responses to pathological conditions disturbing the balance; the stability of the erythrocyte; hæmoglobin and iron metabolism; the plasma and blood volume; the peripheral blood as a pathological index.

Tuesday, April 8, 1930.

The blood and hæmopoietic tissues (continued); the interpretation of specific diseases of the blood; secondary and primary anæmias; *polycythæmia rubra*; the leucocythæmias; the spleen and anæmias; tumour relationships.

Tuesday, April 15, 1930.

Inflammatory and degenerative conditions of the arteries; anatomy; changes with age; the incidence, pathogenesis and ætiology of arteriosclerosis; fatty deposits, atheroma and calcification; hyaline and amyloid degeneration; the arteries in diabetes; productive endarteritis; mesarteritis; *thromboangiitis obliterans*; purulent arteritis; *periarteritis nodosa*; syphilitic arteritis; aneurysms.

Tuesday, May 6, 1930.

Bone; origins and relationships of the tissue; conditions of development and maintenance; calcium and phosphorus; endocrines; vitamins; pathological bone formation and resorption; histology, fractures and repair; non-union; acute osteomyelitis and periostitis.

Tuesday, May 13, 1930.

Bone (continued); chronic osteomyelitis and osteitis; tuberculosis; syphilis; rickets and infantile scurvy; the osteomalacias, *osteitis fibrosa* and *osteitis deformans*; age, endocrines and diet; tumours of bone.

Tuesday, May 20, 1930.

Cardiac pathology; (*a*) the myocardium, the blood supply and arterial disease, atrophy and degeneration, the ætiology and consequences of parenchymatous and interstitial myocarditis, acute and chronic, the conducting system of the heart and pathological lesions; (*b*) endocarditis, ætiology, pathogenesis and bacteriology.

Tuesday, May 27, 1930.

The liver; structure, blood supply and lymphatic connexions in relation to function and the localization of the effects of circulatory disturbances, intoxications and infections; hæmorrhage, degenerations, focal necrosis and suppuration; regenerative power of liver tissue; acute yellow atrophy and the cirrhoses; tumours and tumour dissemination.

Tuesday, June 10, 1930.

The liver (continued); pathological physiology; experimental pathology; carbohydrate, protein, fat and choles-

terin metabolism; detoxication of blood; ascites; bile formation.

Tuesday, June 17, 1930.

Diseases of the mammary gland; structure and functional relationships; lactation and involution; acute and chronic mastitis; precancerous changes; the diagnosis of innocent and malignant tumours; Paget's disease; fat necrosis.

Tuesday, June 24, 1930.

The ovary; development, structure and function and relation to pathology; involution; oophoritis, acute and chronic; cysts and tumours, teratomata, follicular, luteal and blood cysts, endometrioma and carcinoma; parovarian inflammatory and other cysts; connective tissue tumours; secondary growths; the complications and surgical importance of these conditions.

SOME FORTHCOMING COURSES.

THE *Dozentverein für Ärztliche Ferienkurse zu Berlin* (tutorial society for holiday courses in medicine in Berlin) announces permanent courses lasting a fortnight or a month in all branches of medical work and longer post-graduate courses for medical practitioners desiring to undertake practical work under systematic supervision in recognized clinics, hospitals and laboratories. These practical courses last for two or three months.

In October, 1930, there will be a practical laboratory course, a course in diseases of children, a course in general medicine and a course in ophthalmology. In addition there will be a course in industrial hygiene, in insurance work and in medical jurisprudence.

The March course is more extensive. It comprises special instruction in diagnosis and therapeutics, in dermatology and venereal diseases, in obstretrics and gynæcology, in radiology, in general medicine and in general surgery.

The courses are in German, but several of the lecturers are prepared to discourse in English, French or Spanish. Full information can be obtained by writing to the *Sekretär, Kaiserin Friedrich-Haus für das ärztliche Fortbildungswesen*, Berlin, N.W.6, Luisenplatz 2-4.

A special post-graduate course in ophthalmology will be held in Vienna under the auspices of the American Medical Association of Vienna from October 1 to December 5, 1930. The lectures and demonstrations will take place at the I and II Eye Clinic of the *Allgemeines Krankenhaus*. The course will be under the charge of Professor E. Fuchs, Professor J. Meller and Professor K. Lindner. Professor Kolmer will deal with comparative ophthalmology, Professor Schüller will handle radiology, Professor Hirsch will lecture on the pituitary body and the sinuses, while other teachers will deal with radium treatment, special operations, embryology and so forth. The course has been arranged in a systematic manner, so that the whole subject can be covered in the allotted time. A preliminary knowledge of ophthalmology is presupposed. The entire course will be in English. The classes will comprise not less than ten and not more than sixteen graduates. The fee for the course is $250. A certified cheque for fifty dollars or its equivalent should be forwarded to Professor A. Fuchs, Vienna, VIII, Skodagasse 13, together with the application for admission. Applications are accepted in order of priority.

Obituary.

ARCHIBALD PRIMROSE ANDERSON STUART.

IT is with great regret that we have to announce the death of Surgeon-Lieutenant Commander Archibald Primrose Anderson Stuart. While on service in the West Indies he became infected with tuberculosis. The infection was a very virulent and acute one. He was invalided home, but the disease made rapid strides and he died at Deal on February 16, 1930, a few weeks after the onset.

Archibald Primrose Anderson Stuart was the eldest son of the late Thomas Anderson Stuart, the founder of the Medical School of the University of Sydney. He was born in Sydney in 1894. He entered the Medical School at the University of Sydney just before the war, but he enlisted in 1915 and served for three years in the Army Service Corps. He was invalided home in 1918. Two years later he graduated in medicine and surgery. In 1921 he held the appointment of resident medical officer at the Lewisham Hospital and in 1923 he obtained a commission as a surgeon-lieutenant in the Royal Navy. For a time he served in the East Indian Squadron. Later he was transferred to the North Sea Fleet and still later he served in the West Indian Squadron. He is remembered by his contemporaries as a versatile and competent practitioner, a charming companion and a man of much strength of character and initiative. The sympathy of the medical profession is extended to his mother, Lady Anderson Stuart, and to his brothers, one of whom, Dr. B. P. Anderson Stuart, is a well known medical practitioner, specializing in radiology in Sydney.

Correspondence.

A MATTER OF SPELLING.

SIR: When 90% or more medical practitioners adopt a certain way of spelling a certain word, one begins, in one's own mind, to doubt whether the correct spelling of such word is not of the nature of a fad.

Can you inform your readers if albumin should always be used instead of albumen?

Yours, etc.,
"A.B.C."

February 14, 1930.

[Albumen means either the white of egg or the proteid matter contained in white of egg. Albumin is a chemically pure substance. Our correspondent places himself in an untenable position when he suggests that incorrect spelling, if widely used, should be adopted. We endeavour to follow the best authorities in regard to orthography and typography. If accuracy is a fad, THE MEDICAL JOURNAL OF AUSTRALIA wishes to be regarded as a faddist.—EDITOR.]

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Main, Arthur Wareham, M.B., Ch.M., 1924 (Univ. Sydney), Roma.

Beith, Bruce McNeil, M.B., Ch.M., 1916 (Univ. Sydney), Brisbane.

Barkley, Alfred Oswald, M.B., Ch.M., 1923 (Univ. Sydney), Atherton.

Restoration to the Register:

Ellot, Henry Pritchard, M.B., C.M., 1894 (Univ. Edinburgh), Cooktown.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1912 and 1915, of New South Wales as duly qualified medical practitioners:

Breslin, Francis Louis, M.B., Ch.M., 1922 (Univ. Sydney), Burns Bay Road, Lane Cove.

Powell, Arthur Hunter, M.B., B.S., 1912 (Univ. Melbourne), Armidale.

Webb, Arthur Liddon, M.B., B.S., 1922 (Univ. Adelaide), F.R.C.S. (England), 1929, Wentworth Falls.

For additional registration:

Thomas, Alfred Charles, F.R.C.S. (Edinburgh), 1929.

Corrigendum.

OUR attention has been drawn to the fact that an error was made in the initials of the author of an article entitled: "A Phenomenon in Anæsthesia," published in the issue of June 29, 1929, at page 866. The author was Dr. W. P. Kelly.

Books Received.

A MANUAL OF PHARMACOLOGY, by Walter E. Dixon, M.A., M.D., B.S., B.Sc., D.P.H., F.R.S.; Seventh Edition; 1929. London: Edward Arnold and Company. Demy 8vo., pp. 496, with illustrations. Price: 18s. net.

RECENT ADVANCES IN PREVENTIVE MEDICINE, by J. F. C. Haslam, M.C., M.D. (Edinburgh), M.R.C.P. (Edinburgh), D.P.H., with a chapter on the Vitamins by S. J. Cowell, M.A., M.B., M.R.C.P.; 1930. London: J. and A. Churchill. Post 8vo., pp. 328. Price: 12s. 6d. net.

Diary for the Month.

MAR. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 4.—New South Wales Branch, B.M.A.: Ethics Committee.
MAR. 4.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
MAR. 5.—Victorian Branch, B.M.A.: Branch.
MAR. 6.—South Australian Branch, B.M.A.: Council.
MAR. 7.—Queensland Branch, B.M.A.: Branch.
MAR. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 13.—Victorian Branch, B.M.A.: Council.
MAR. 13.—Surgical Section, Queensland Branch, B.M.A.

Medical Appointments.

Dr. C. R. Dunkley (B.M.A.) has been appointed Deputy Quarantine Officer, Fremantle.

* * *

Dr. Andrew Norman Dickson (B.M.A.) has been appointed Certifying Medical Practitioner at Lismore, Victoria.

* * *

Dr. Jack Blakely Street (B.M.A.) has been appointed Government Medical Officer at Moruya, New South Wales.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

MELBOURNE HOSPITAL, MELBOURNE, VICTORIA: Honorary Surgeon to Out-Patients.

THE ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED), SOUTH AUSTRALIA: Temporary Honorary Assistant Surgeon.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Vacancies.

THE PUBLIC SERVICE BOARD, NEW SOUTH WALES: Medical Officer (female).

VICTORIAN EYE AND EAR HOSPITAL, VICTORIA: Honorary Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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